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Table of Contents.

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ORIGINAL ARTICLES— Page.

A Tuberculosis Survey of Children in a Provincial City, by K. G. Kerr .. 901
Hydatid Disease of Vertebræ, by Leslie J. Woodland 904
Prolapse of the Nucleus Pulposus in the Cervical Region, accompanied by Compression of the Spinal Cord, with Report of a Case, by James Isbister .. 910

REPORTS OF CASES—

Report on a Case of Cerebro-Spinal Meningitis due to Cryptococcus Histolytica (Torula Hominis), by Lawrence W. Alderman .. 914

REVIEWS—

Pharmacology in Practice .. 916
Role of Heredity and Hormones in Sex .. 916

LEADING ARTICLES—

The Sins of Medicine .. 917

CURRENT COMMENT—

The Fate of Substances Injected Intrathecally .. 918
Prognosis of Acute Myocardial Infarction .. 919

ABSTRACTS FROM MEDICAL LITERATURE—

Surgery .. 920

MEDICAL SOCIETIES— Page.

Melbourne Pædiatric Society .. 922

CORRESPONDENCE—

The Painful Stiff Shoulder .. 925

POST-GRADUATE WORK—

The Melbourne Permanent Post-Graduate Committee .. 926
The Post-Graduate Committee in Medicine in the University of Sydney .. 926

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA .. 927

AUSTRALIAN MEDICAL BOARD PROCEEDINGS—

Queensland .. 928

NOMINATIONS AND ELECTIONS .. 928

CORRIGENDUM .. 928

MEDICAL APPOINTMENTS .. 928

DIARY FOR THE MONTH .. 928

MEDICAL APPOINTMENTS: IMPORTANT NOTICE .. 928

EDITORIAL NOTICES .. 928

A TUBERCULOSIS SURVEY OF CHILDREN IN A PROVINCIAL CITY.[1]

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A SMALL TUBERCULIN SURVEY of country people was reported by Anderson in 1940 (Anderson, 1940*b*), and he concluded by saying: "Perhaps this small study will stimulate medical men in country centres to make tuberculin surveys. From such surveys it is likely that information of much interest and value would be forthcoming."

In anticipation of a visit to Bendigo by the Department of Health Chest X-Ray Survey Staff in November, 1948, it was decided in August, 1948, to make a beginning with tuberculin testing of children, so that X-ray examination of those under fourteen years of age might be confined to the positive reactors and that family investigation in the case of reactors might be made. At the same time, it was felt that the testing of as many adults in industry as possible would be useful, in order to ascertain the degree of tuberculous infection in the community, compared with surveys which have been made in other communities.

In 1934 Gregory Kayne and B. Weill-Hallé referred to the "importance of tuberculin tests in infancy and childhood (*a*) in the diagnosis of tuberculous disease, (*b*) in dealing with home contacts, (*c*) in connexion with B.C.G. vaccination". I should add "(*d*) as an indicator of a possible family source of infection". In regard to mass testing under Victorian conditions, especially in the country, with relatively few children who show primary lesions in a state of activity, probably (*d*) should come first—that is, as an indicator to adult "open" cases.

Bendigo is a provincial city, which, together with the Borough of Eaglehawk, has a population of 30,800. It stands intermediate between rural areas and highly urbanized capital cities, but it is distinguished by a relatively high death rate from pulmonary tuberculosis over many years. The mining industry is responsible for many deaths from silicosis, often complicated by tuberculosis. It was expected that percentages of positive reactors in all age groups would be relatively high.

In December, 1947, the children in two orphanages had been tested by the Vollmer patch method. There were 159 children aged from two to sixteen years, and only seven reacted (4·4%).

In this survey, the Mantoux intradermal test was used for school children and adults, and the Vollmer patch test for infants and pre-school children. For the intradermal tests, Commonwealth Serum Laboratories old tuberculin made from human strains was used in a dilution of 1:1000 and 0·1 millilitre was injected into the left forearm. Readings were generally made in forty-eight hours, very occasionally between three and five days. The tuberculin was used within two to three weeks of issue from Parkville and stored meanwhile in the refrigerator at the Health Laboratory, Bendigo, until the day of use.

In 1940, Wunderly used this technique with Commonwealth tuberculin as recommended by a committee of the National Health and Medical Research Council. He compared results with freshly diluted tuberculin and a proprietary form of tuberculin, also with tuberculin P.P.D. of two makes, and considered that the advantage was with the Commonwealth product even diluted up to three months previously.

Record cards issued by the Commonwealth Department of Health for use in connexion with B.C.G. vaccination

[1] Read at a meeting of the Melbourne Pædiatric Society on August 10, 1949.

TABLE I.
Tuberculin Tests of 7682 Persons, Bendigo, 1948.

Age.	Males.			Females.		
	Number Tested.	Number of Reactors.	Percentage of Reactors.	Number Tested.	Number of Reactors.	Percentage of Reactors.
Pre-school:						
1 to 5 years	482	4	0·83	492	9	1·8
School:						
Grades 1 and 2 (5 to 7 years)	767	18	2·3	660	12	1·8
Grades 3 and 4 (8 and 9 years)	491	12	2·4	485	20	4·1
Grades 5 and 6 (10 and 11 years)	555	28	5·04	495	20	4·04
Forms I and II (12 and 13 years)	532	28	5·2	488	31	6·3
Forms III and IV (14 and 15 years)	248	15	6·04	254	18	7·08
Forms V and VI (16 and 17 years)	44	2	4·5	63	3	4·7
Industry:						
15 to 20 years	178	25	14·4	385	49	12·7
21 to 25 years	169	49	28·9	122	32	26·2
26 to 30 years	124	61	49·2	36	10	27·7
31 to 35 years	111	61	54·9	12	6	50·0
36 to 40 years	140	88	62·8	14	7	50·0
41 to 45 years	108	67	62·04	13	7	53·8
46 to 50 years	93	62	66·6	7	4	57·1
51 to 60 years	112	85	75·9	2	2	100·0
Totals	4154	—	—	3528	—	—

refer to tuberculin tests as being carried out with 0·1 millilitre of 1:1000 old tuberculin. In 1942, Darcy Cowan made reference to tests on nurses at the Royal Adelaide Hospital being made with 1:1000 Commonwealth old tuberculin. He stated that "a tuberculin test is indispensable in a survey of children for tuberculosis". In 1947, Lowe and Hallam reported on "tuberculous infection in male medical students" after tests with the same tuberculin.

In the investigation of a series of 84 nurses in Bendigo given Canadian or Commonwealth B.C.G., I have found 87% conversion as estimated with 1:1000 Commonwealth tuberculin.

With a mass survey, tests with stronger dilutions can be made when further investigation is being carried out of those suspected on radiological grounds of having active disease.

Degrees of reaction to both intradermal and patch tests were classified—in the former according to the classification of the Henry Phipps institute, as quoted by Wunderly (1940), and in the latter according to Vollmer, as quoted by Colebatch (1941).

A raised red area of five millimetres in diameter was taken as the minimum positive reaction. All grades of reaction were encountered. Of the 7682 persons tested, 6056 were children under or of school age. Table I and Figure I show the results of these tests.

It is surprising and encouraging to find such a generally low percentage of positive reactors in most age groups in a city which, in the five-year period 1941 to 1945, had a death-rate from pulmonary tuberculosis of 5·9 per 10,000 of population, as compared with rates of 4·1 for Melbourne, 4·0 for Ballarat and 3·5 for Geelong. Figure II shows the consistently high rate for the last fifty years in comparison with the rate for the other cities.

Children are remarkably free of evidence of primary infection, ranging from 1% with a positive reaction under the age of five years to 7% at fifteen years. Compare these figures with those of Anderson (1940*a*) for children in Sydney in 1940—6% among children under four years, 12% among those under nine years, and 21% among those from ten to fourteen years, P.P.D. and the one injection technique being used.

Fry (1945), in Adelaide, found 2% of positive results among children of three to seven years, 6% among those of eight to ten years, 9% among those of eleven to thirteen years, and 8% among those of fourteen to sixteen years, and he claimed that the over-all percentage of reactors among children in the survey, with the use of both 1:1000 and 1:100 dilutions of Commonwealth tuberculin, was among the lowest recorded.

Myers (1948) states that in 277 schools Jordan found among those tested not one infected child in 219, that,

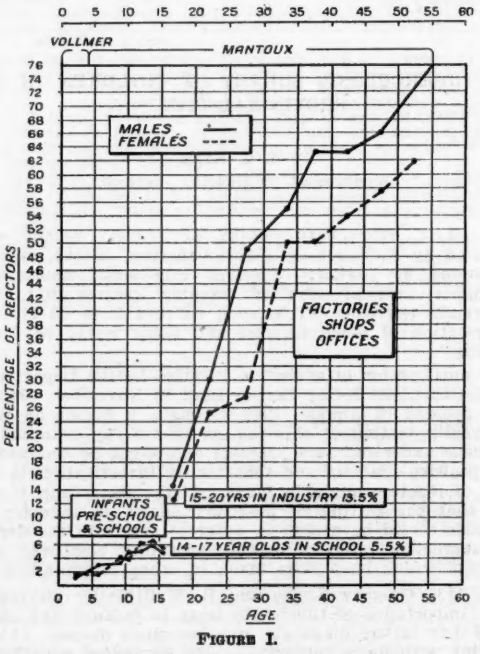

FIGURE I.

among the senior students in high schools of entire counties, the incidence of tuberculin reactors is 5% to 8%, and that in the largest city 12·5% of high school students react to tuberculin. He claims that tuberculosis eradication has been accomplished up to the age of fifteen years in some areas.

Bendigo figures appear to compare very favourably with those quoted. Is it too much to suggest that the existence of a Tuberculosis Bureau since 1929 and a chalet of 24 beds since 1933, with constant education of patients by a visiting nurse in proper disposal of sputum, has been a factor in reducing the incidence of tuberculous infection among children? During the last ten years there have been only four deaths of children from tuberculous meningitis.

There is a sudden and apparently significant jump from 5·5% at the age of fourteen to seventeen years (school children of both sexes) to 13·5% at the age of fifteen to twenty years (in industry) and 27% at the age of twenty-one to twenty-five years. There appears to be a significant increase in the chances of first infection when a child from school first enters industry. This increase is probably more real than apparent as there are individuals aged fifteen, sixteen and seventeen years included in both groups.

There were thus 220 positive reactors among children under or of school age. Those of fourteen years and over, 38 in number, underwent X-ray examination of the lungs with a microfilm, and I have no record of the findings, but for those under fourteen years a large film was used.

Allen (1949) refers to the statement of Myers and Harrington that "X-ray examination fails to locate the lesion of first-infection type in 75% or more of children who react to the tuberculin test, and usually in the 25% in which a lesion is located no clinical significance is attached to it".

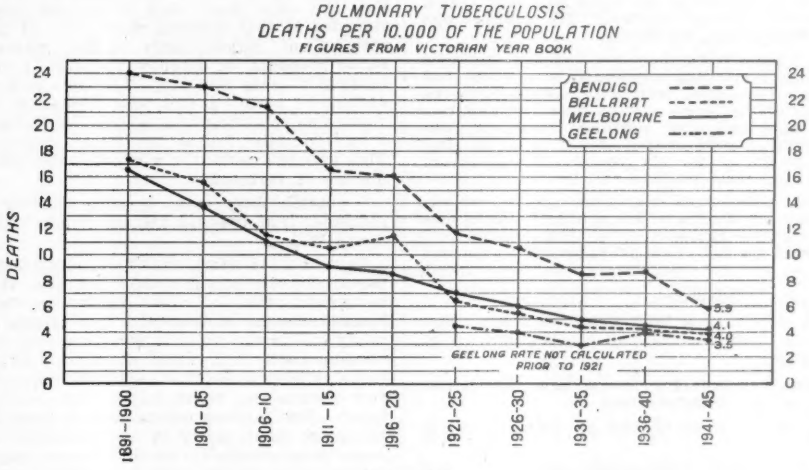

FIGURE II.

I am indebted to Dr. Lyell Andrews, Director of Mass Radiography, Department of Health, Victoria, for radiological reports on the large films. Some of the children underwent X-ray examination privately, but of 148 examined (all positive reactors) the lung appearances of 122 were considered to be within normal limits. In 24 instances there were calcified nodes, calcified hilar glands, fibrotic markings, peribronchial infiltration or an increase in broncho-vascular markings. One child had a "doubtful right apex" and another a small shadow in the subapical region of the right lung. Neither of these, on further investigation, was considered to have an active lesion. No organisms were grown from the fasting gastric contents of these two patients. One child, a boy of thirteen years, had a history of a tuberculous spine; a girl of fourteen years developed a tuberculous hip during the next few months; a girl of seven years had been in the Austin Hospital with a primary lesion and a positive result from gastric culture, but this was now healed and she was well.

Only one child, a girl of nine years, who was known to have been "tuberculin-positive" for two years, had an active lesion in the lung. This appears to have been allergic in type and resolved after six months' treatment. Attempted culture of organisms from fasting gastric contents was unsuccessful in this case.

I quote from the report of the Prophit Survey:

> In the past it has been insufficiently realised that, with the exception of infants in the first year of life, the greatest risk in tuberculous families is incurred, not by the children, but by the young adult members of the family.

For years, tuberculosis bureaux in Victoria have been concentrating on the regular examination of contacts, especially young adults, for the purpose of case-finding. It appears that tuberculin testing and regular examination of young adults in industry are going to pay more dividends in this way than the X-ray examination of child reactors.

Nevertheless, the child reactor is a useful pointer towards possible adult cases, as I shall show later.

I should like to retest all these children, both those giving positive and those giving negative results, every twelve months until they reach early adult life. In the absence of further doses of infection, will the positive reaction remain? It appears, in elderly people, that, even in the presence of pronounced radiographic evidence suggestive of past tuberculous infection, the allergic state can have been lost, and there will be no signs of activity.

Is it preferable to have overcome primary infection in childhood, rather than to encounter it in early adult years, in order to develop increased resistance against the secondary phenomena of adult pulmonary tuberculosis? We do not know whether these early infected children are more likely or less likely than uninfected children to be suffering from adult pulmonary tuberculosis in ten or twenty years.

Preventive work among nurses suggests that the reactors are in the better position. In that case, our main problem should be to watch the "tuberculin-negative" adolescents and be on the lookout for conversion of the tuberculin reaction. The administration of B.C.G. to these "tuberculin-negative" adolescents is indicated.

This is the paradox—that it appears to be safer for a child to be primarily infected, if infection does not extend beyond the healed primary stage, than to be virgin soil at puberty or in early adult years. But it is our duty to prevent infection of those who are free and reinfection of the already infected.

But why is the five to fifteen years age group apparently so resistant to infection?

A trained tuberculosis nurse visited the homes of the 220 reacting children. She took a family history and arranged for X-ray examination of adult members of the family. In relation to 81 of the children she found 70 persons with pulmonary tuberculosis who had been intimately associated in the home as probable sources of infection during the lives of the children; in some instances several children were related to the one infected person, and sometimes one child had been in contact with several infected persons in the family.

Of these seventy persons with tuberculosis, all but five had already been registered with the Tuberculosis Bureau. Thus, five new cases of active pulmonary tuberculosis were discovered as a result of the investigation, in four of which *Mycobacterium tuberculosis* was present in the patient's sputum.

In addition, in relation to nineteen more reactors, there were twenty more subjects of pulmonary tuberculosis with very probable contact in the home.

The five new cases were as follows.

CASE I.—J.B., a male, aged forty-one years, had had a cough for three months with blood-stained sputum, loss of energy and loss of weight. A radiograph of the lungs showed tuberculous infiltration with a cavity in the upper zone of the right lung.

CASE II.—M.B., a female, aged forty-seven years, had a history of "an abscess on the lung" eighteen years ago when she was told that there was "a thickened ring" showing in the right lung radiographically. She stated that she was well and had no cough or sputum. However, a radiograph showed scattered fibro-calcareous infiltration in the upper half of the right lung with well-marked cavitation in the right infraclavicular region, and fairly extensive fibro-calcareous nodules in the upper half of the left lung with scattered calcification in the lower half. The result of examination of sputum was positive.

CASE III.—H.P., a male, aged thirty-six years, had had a cough for two years. Radiographically, the upper zones of both lungs were found to contain a soft-looking infiltration of considerable extent. In the left subapical region there was a large thin-walled cavity and in the right subapical region were probably several smaller cavities. The sputum was found to contain tuberculosis organisms.

CASE IV.—M.P., a male, aged forty-five years, stated that he was well, but had a morning productive cough. He had hæmoptysis two years ago and "bronchitis" eighteen months ago. A radiograph of the lungs showed fibrotic infiltration at the right lung apex and faint mottling throughout both lung fields suggesting a mild degree of silicosis. He had been a grinding machinist for six years. The result of examination of sputum was positive.

CASE V.—P.B., a male, aged thirty-one years, an ex-serviceman, had tubercle bacilli in his sputum. He is now under treatment by the Department of Repatriation and other clinical findings are not made available to the Bendigo Bureau.

Summary.

In a provincial city with a high death rate from pulmonary tuberculosis, the percentage of tuberculin reactors among children is low.

Entry of children into industry results in a sudden rise in the percentage of tuberculin reactors.

Only one child, of 220 reactors, had an active lesion in the lung.

Five new cases of adult tuberculosis were discovered as a result of tuberculin testing of children.

Conclusions.

1. In many cases, children react to tuberculin because there has been a definite family source of infection.

2. Tuberculin tests in children appear to be a reliable indicator as to where to find cases of pulmonary tuberculosis.

3. In the past, the Tuberculosis Bureau has been able to discover and register the majority of active cases in the community, but skin testing of children is a useful additional method of case finding.

References.

Allen, F. M. B. (1949), "Some Practical Problems of Tuberculosis in Childhood", *British Medical Journal*, Volume I, page 747.

Anderson, Douglas (1940*a*), "Studies in Tuberculosis. I. The Incidence of Tuberculosis Infections in and about Sydney, New South Wales, as Shown by the Intracutaneous Tuberculin Test", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, page 747; (1940*b*), "Studies in Tuberculosis. IV. The Incidence of Tuberculosis Infection in Country People in New South Wales Compared with that in City Dwellers", *ibidem*, Volume I, page 829.

Colebatch, J. H. (1941), "The Tuberculin Patch Test", THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, page 640.

Cowan, D. R. W. (1942), "Pulmonary Tuberculosis: The Adelaide Health Survey", THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, page 353.

Fry, H. K. (1945), "Adelaide Local Board of Health Survey of School Children by Mantoux and Patch Test", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, page 604.

Kayne, G. G., and Weill-Hallé, B. (1934), "Contribution to Comparative Study of Tuberculin Tests in Infants and Children" *British Medical Journal*, Volume II, page 464.

Lowe, T. E., and Hallam, K. H. (1947), "The Incidence of Tuberculous Infection in Male Medical Students in Melbourne", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, page 749.

Myers, J. A. (1948), "Eradication of Tuberculosis by Epidemiological Methods", *American Journal of Public Health and The Nation's Health*, Volume XXXVIII, page 516.

Wunderly, H. (1940), "An Investigation into the Incidence of Pulmonary Tuberculosis in Young Women of Adelaide in the Age Group Fifteen to Thirty Years", THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, page 229.

HYDATID DISEASE OF VERTEBRÆ.

By LESLIE J. WOODLAND,
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AT the Royal Prince Alfred Hospital, during the past four years, I have seen three patients with hydatid disease of vertebræ; one patient was admitted under my care, the others were seen in consultation. When I examined the records of patients with hydatid disease of bone admitted to the Royal Prince Alfred Hospital since 1910, I found that the only three cases of hydatid disease of vertebræ recorded at that hospital were those of the three patients seen by me. Subsequently, by the courtesy of the Medical Superintendent of Sydney Hospital, I found from their medical records that since 1922 two patients with hydatid disease of vertebræ had been treated at Sydney Hospital. I am also reporting another case of hydatid disease which I have encountered at the Royal Prince Alfred Hospital. This patient's condition was originally classified as hydatid disease of vertebræ.

I publish these cases because of their rarity and as a reminder that hydatid disease of bone still occurs in New South Wales.

Dew (1928*a*) states that hydatid disease in bone is comparatively rare and is always primary. It is now generally recognized that all the differences detectable between hydatid disease of bone and that of any other tissues are solely due to the peculiar structure and resistant nature of osseous tissue. Hydatid disease of bone differs in two respects from hydatid elsewhere in that exogenous vesiculation occurs and there is an absence of an adventitious layer. The embryo commences to grow in the cancellous tissue or more rarely in the medullary canal. Although some pressure absorption of the bone may occur with dilatation of the bony space in which the parasite lies, this occurs so slowly that the parasite begins to grow in the line of least resistance, insinuating itself by microscopic prolongations of both its layers along the bony canals. Owing to the restrictions imposed upon their growth, such processes do not contain fluid, but, as they enter wider canals, or as bony absorption leads to the formation of small cavities in the bone, localized swellings may appear. It is usually found that so-called cysts, which are able to form, are almost solid masses of hyaline material surrounding central nuclear material, and, in appearance, they resemble granules of boiled sago. As a rule, any small cysts that form lie in actual contact with bone or cellular debris, and have only a partial cellular envelope. Pressure on and occlusion of small arteries supplying the bones occur; hence, sooner or later, necrosis of the older more centrally placed portion of the lesion takes place. When the periosteum has been perforated the parasite invades the soft tissues and immediately takes on a typical univesicular type of growth, producing fluid and reproductive elements and stimulating the formation of a fibrous adventitia. The extra-osseous extensions may be very extensive. Hydatid disease of bone is an extremely chronic disease.

Hydatid disease of the vertebral column may commence in the bodies, pedicles or laminæ and spread in the cancellous tissue in the same way as in other bones. The slow microscopic infiltration of hydatid elements does not present the appearance, seen in carcinoma or sarcoma of bone, of osseous structures being completely dissolved away; neither does hydatid infiltration approach in the slightest degree the intensity of lymphocytic erosion seen in Pott's disease, or the toxic osteolysis seen in pyogenic

spondylitis. Dew (1928*b*) states that usually the disease remains confined to bone for years, but, sooner or later, extra-osseous extension takes place. Very often the extension affects the posterior vertebral muscles and fairly large multiple cysts may develop in them. If any hydatid is detected near the spine, the possibility of extra-osseous extension of cysts of the vertebræ must always be considered. Cysts may extend from the vertebræ into the mediastinum, the retroperitoneal tissues or the pelvis, but the most serious result is the formation of cysts within the spinal canal, with consequent pressure on the cord. Cysts which extend into the extra-dural space are always multiple. Dew (1928*c*) states that the prognosis in vertebral hydatid disease is almost hopeless as regards complete cure. Subsequent to an operation, there is a risk of severe post-operative hæmorrhage, which may be fatal as in a case reported by Corlette (1920) of massive cyst of the ilium. A further risk is the development of sepsis in a large cavity, or cavities, which frequently causes death. If the immediate post-operative risks of hæmorrhage and sepsis have been overcome, recurrence is the usual fate, but symptoms of recurrence may be delayed for years.

Investigation of the records of the Royal Prince Alfred Hospital shows that in the period from 1910 to 1948, 852 patients with hydatid disease were admitted to hospital. Of the admissions, 16 were for hydatid disease of bone. These admissions represent 11 patients, as four patients were admitted on more than one occasion. The admissions for hydatid disease of bone represent 1·9% of the total admissions. The scapula was involved in one case, ribs in two cases, the femur in one case, the innominate bone in three cases, the humerus in one case, and vertebræ in three cases.

Investigation of the records at Sydney Hospital shows that for the period from 1922 to 1942, 205 patients were admitted to hospital with hydatid disease. Of the admissions seven were for hydatid disease of bone. These admissions represent four patients, one patient having been admitted on four occasions. The admissions for hydatid disease of bone represented 3·5% of the total admissions. One patient had hydatid disease of the ilium. Another patient had an ununited fracture of the upper end of the femur, due to hydatid disease. There were two patients with hydatid disease of vertebræ.

Examination of the Louis Barnett Hydatid Registry of the Royal Australasian College of Surgeons showed that the total number of cases of hydatid disease recorded in the registry up to 1948 was 1874. Of these, the total number of cases involving bone was as follows: Australian cases, 31; New Zealand cases, 17; Total, 48; that is, 2·4% of the total number of cases of hydatid disease recorded in the registry occurred in bone. The number of cases involving vertebræ, as recorded in the registry, was as follows: Australian cases, 17; New Zealand cases, 7; total, 24; that is, 50% of the number of cases of hydatid disease in bone occurred in vertebræ.

The frequency of hydatid disease of bone, quoted by Dew (1928*d*) has been given by various observers as follows: Thomas, 1·6%; Vegas and Cranwell, 0·59%; Dévé, 0·9%; Mills, 1·0%; Vinus, 1·29%. The total number of cases involving bone recorded in the Louis Barnett Hydatid Registry represents a frequency of 2·24. In the combined series of admissions of patients with hydatid disease, taken by me from the records of the Royal Prince Alfred Hospital and Sydney Hospital, the percentage of admissions for hydatid disease of bone was 2·1%. The common sites of infestation are the vertebræ, the pelvis, the femur and the humerus. In the Hydatid Registry the vertebræ are the most frequently affected bones, representing 50% of cases. Bellini (1946) states that osseous involvement occurs in only a small proportion of echinococcus infestations, probably about 1%. The spongy tissue of the vertebral bodies is a favourite site of hydatid disease. Nearly 50% of all cases of osteohydatidosis involve the spine, with the bony pelvis following in order of frequency. In the series under review the vertebræ were affected in 33% of patients with hydatid disease of bone.

The infestation, as with hydatid disease elsewhere, usually commences in childhood; but, whilst it is confined to the bone, the growth of the hydatid is so slow that symptoms do not occur before adult life. In the five cases under review of hydatid disease of vertebræ, the average age of the patients when symptoms commenced was thirty-four years. Symptoms do not occur until the destruction of the bone leads to fracture or dislocation, or until the hydatid cyst has erupted from the bone and caused pressure or obvious swelling. In four of the five cases here reviewed, the patients had symptoms of pressure on the spinal cord. Four of the five patients died, the average duration of life after the onset of symptoms being eleven years. Of the patients who died, three died of sepsis in an operation wound or in the bladder; one died from misadventure. The original site of infestation in three cases was the upper lumbar vertebræ and in two cases the mid-thoracic vertebræ.

Reports of Cases.

CASE I.—The patient, a female, aged fifty-five years, was admitted to the Royal Prince Alfred Hospital on September 16, 1946. She had lived in western New South Wales all her life and was perfectly well until four years before admission, when she first noticed that, whilst stooping to pick up objects from the floor, she experienced sharp pain in the left lower lumbar region. This shooting pain at first lasted only whilst she was bending, but later would persist for several hours. The pain radiated into the front of the left thigh. After a course of gold injections given in the country the patient said that the pain was less for some months, but then returned. During the year prior to admission to hospital she had noticed that the pain became worse when she walked; and during the last three months of the year the pain had intensified and was unrelieved by rest. When the patient was straining at stool the pain in the back was very severe. She was rested in bed in a country hospital for two months before being admitted to the Royal Prince Alfred Hospital.

On examination of the patient it was noticed that the normal lumbar lordosis was absent. Slightly increased prominence of the spinous process of the second lumbar vertebra was noted with a sharp lateral angulation at the same level and convexity to the right. On the convex side of a compensatory uniform thoracic scoliosis to the left, there was present a diffuse swelling on the side of the thoracic vertebræ, extending from about the third thoracic vertebra to the upper part of the lumbar region. Movements of the spine were restricted in all directions. There was guarding of the lumbar sacro-spinal muscles. Reflexes and sensation were normal. X-ray examination (see Figures I and II) showed increased density and alteration of the cancellous architecture of the bodies of the first, second and third lumbar vertebræ. Pronounced wedging of the body of the second lumbar vertebra on the left side and moderate wedging of the third lumbar vertebra on the right side, with slight subluxation to the right of the spine above the second lumbar vertebra, had occurred. Light flakes of calcification were seen beyond the limits of the vertebræ.

It was considered that the patient was suffering from Pott's disease of the spine with light calcification in a paravertebral abscess. The possibility of hydatid disease of the vertebræ was completely overlooked. The diffuse swelling over the convex side of the thoracic scoliosis was thought to be a deposit of fat.

On October 20, 1946, operation was commenced with the intention of performing a spinal fusion. After the interspinous ligament between the second and third lumbar vertebræ had been split caseous material was found. It was still thought that this was a tuberculous abscess until small daughter cysts, which resembled sago granules, were seen. A large quantity of hydatid debris and daughter cysts was evacuated; it was found to come from a very extensive paravertebral cavity, which extended forward, laterally and distally on the left side of the mid-line. It was not possible to touch the bottom of the cavity with the tip of a sucker, because of the depth of the cavity. Penicillin-sulpanilamide powder was placed in the wound, and, on Professor H. R. Dew's suggestion, a drainage tube was left in for twenty-four hours. The patient stated that the operation completely relieved her pain. The debris removed from the cyst at the time of the operation was found to be sterile on incubation. The diffuse, longitudinal swelling on the left side of the thoracic part of the spine increased and on November 13, 1946, this swelling was drained. A large quantity of hydatid material was evacuated from a cyst, which extended, deep to the sacro-spinalis muscle, from about the second lumbar vertebra to the second thoracic vertebra on the left side. The patient commenced to run a high temperature; obviously the extensive hydatid cysts were infected in spite of prophylactic and therapeutic penicillin injections, which reduced the temperature for a time. Later, cultures of *Bacillus coli*

communis and *Proteus vulgaris* were grown from the discharge. The patient's condition gradually became worse and she died on February 18, 1947.

Examination *post mortem* revealed a sinus to the left of the second lumbar vertebra, which was discharging purulent material and small hydatid cysts from a large cavity in the left psoas sheath. Hydatid cysts were scattered beneath the full extent of the left thoracic sacro-spinalis muscle. The second and third lumbar vertebræ (see Figures III and IV) were extensively infiltrated; the intervertebral disks were destroyed to a lesser extent. No cysts were found elsewhere in the body.

This case illustrates the fatal nature of hydatid disease of the bone, when it is impossible to amputate or completely to excise the affected bone, and it also illustrates the widespread growth of hydatid cysts which occurs once the parasites have broken through the restricting bone. In this patient hydatid disease extended through soft tissues from about the second thoracic vertebra posteriorly to the inguinal ligament anteriorly. In this patient (the only one in the series) there was no pressure on the spinal cord. This probably accounted for the delay in diagnosis and the consequent very extensive spread throughout the other extra-osseous tissues. This case also illustrates that, in spite of modern antibacterial therapy, when infection occurs in extensive hydatid cavities, death from sepsis cannot always be prevented.

CASE II.—A man, aged twenty-eight years, was admitted to the Royal Prince Alfred Hospital in 1932 under the care of Dr. Hugh Poate. He gave a history that about two years previously he had noticed a small lump on the right side of his chest just below the scapula, and, at about the same time, the patient developed a cough. The swelling, which was painful at times, gradually increased in size. The lump was excised at Armidale Hospital when it had reached the size of a man's fist. Three months prior to the patient's admission to the Royal Prince Alfred Hospital, the lump recurred at the site of operation, and it rapidly increased in size. On January 12, 1932, the swelling, which was considered to be a tuberculous abscess of the chest wall, was incised. Greenish pus was found to drain extrapleurally around the chest wall beneath the ribs. It was noted that there was necrosis of the seventh, eighth and ninth ribs on their inner aspects. A guinea-pig, inoculated with the pus, did not yield any evidence of tuberculosis. A sinus had persisted ever since. In 1935 kyphosis of the mid-thoracic part of the spine was noticed. In September, 1942, the patient noticed that pain in the mid-thoracic region of the back would follow stooping. He had never been free of pain since then. In 1943 he noticed weakness of both legs, which became worse, and, for six months preceding his readmission to the Royal Prince Alfred Hospital in May, 1945, he had been unable to walk.

On examination of the patient severe spastic paralysis of both lower limbs was found with impaired sensation. A gibbus was present over the sixth, seventh and eighth thoracic vertebræ. Lumbar puncture on May 8, 1945, showed that the cerebro-spinal fluid was clear and the pressure low. The result of the Queckenstedt test was negative. On May 31, 1945, examination of the cerebro-spinal fluid showed that the globulin content was not increased; the protein content was 100 milligrammes *per centum*. X-ray examination (see Figures V and VI) suggested chronic osteomyelitis of the seventh, eighth and ninth ribs on the right side. The corresponding vertebræ showed slight collapse with irregular destruction and increased density. In addition, large cavities were seen in three vertebræ. The X-ray pictures taken in this case were not good films as correct posturing was difficult because of the spinal deformity and the patient's spastic paraplegia. I examined him in consultation and considered that the spastic paraplegia was due to pachymeningitis following chronic osteomyelitis of the vertebræ. On June 7, 1945, Dr. Money performed laminectomy. At about the level of the seventh thoracic vertebra the dura was found to be thickened, the appearance suggesting a spinal meningioma. Further exploration revealed three large cavities in the bodies of the vertebræ. From one cavity, a white lining membrane, resembling a hydatid ectocyst, was removed. From another cavity some gelatinous purulent material was aspirated. The patient recovered from the operation and the wound healed. Pathological examination of a wall of a cavity in the body of one vertebra showed laminated membrane—the wall of a hydatid cyst. The Casoni test, performed after operation, gave a doubtful result. The result of the complement fixation test for hydatid was negative. A further operation was performed in October, 1945, because of pain in the legs and loss of control of the bowels, and because the spastic condition had not become less. Fibrous granulation tissue appeared to be compressing the spinal cord at the level of the sixth and seventh thoracic vertebræ. The granulation tissue was removed until pulsation was observed in the cord. The patient's condition did not improve. He was readmitted to hospital on September 8, 1947. He had spastic paraplegia, complete anæsthesia below the level of the tenth thoracic spinal segment, and an automatic bladder. A fluctuant swelling was present over the right eighth rib in the posterior axillary line. A blood count showed 10,000 leucocytes per cubic millimetre of which 71% were neutrophile cells and 3% eosinophile cells. The Casoni reaction was obtained. The fluctuant swelling, which was an infected hydatid cyst, was incised on September 18, 1947. This procedure relieved most of the patient's pain. Subsequent blood counts showed that the number of leucocytes varied from 12,000 to 14,000 per cubic millimetre and the proportion of eosinophile cells rose to 7%. After a lumbar puncture, the patient developed retention of urine. On September 29, 1947, a further laminectomy was performed, and about twenty small hydatid cysts were found in the extra-dural space at the level of the ninth and

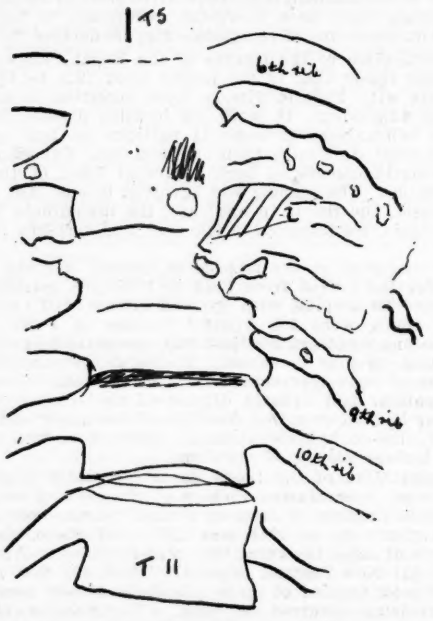

FIGURE V.

Case II (tracing of X-ray film). Antero-posterior view of thoracic spine shows disappearance of joint space between bodies of sixth and seventh thoracic vertebræ and narrow joint spaces between seventh and eighth thoracic vertebræ. Note destruction of ribs on right side with cyst-formation and patchy sclerosis. Some portions of ribs are expanded, other parts eaten away and narrowed. Bony ankylosis due to superadded infection has occurred between sections of seventh and eighth ribs.

tenth thoracic vertebræ. Evacuation of these cysts did not, however, restore pulsation to the spinal cord, which was compressed proximally by scar tissue. Five days later the patient developed generalized urticaria, which was relieved by "Benadryl". He was not relieved by the operation. I have recently heard (June, 1949) from this patient's doctor in the country that the patient died after an accident in a wheel-chair. The patient and the chair were found in a dam at the foot of a hill.

This case illustrates the formation of fairly large cavities in bone; this is rarely seen in hydatid disease of vertebræ. Bellini (1946) states that vertebral hydatidosis shows no tendency to the formation of interosseous cyst-like spaces, which weaken the vertebral body. Coexistent involvement of contiguous ribs is a sign peculiar to vertebral hydatidosis, and an important differential feature. The primary infestation in this case could have been in either a rib or a vertebra. The involvement of a contiguous rib and

vertebra supports Dew's (1928*c*) opinion that, in spite of some statements to the contrary, the articular cartilage does not offer much resistance to the parasitic growth. The intervertebral disks appear to be much more resistant to the disease, but nevertheless it must be via the disks that adjacent vertebræ are affected. For sixteen years this patient had a discharging sinus, the probable cause of which was infection in hydatid infestation of ribs and vertebræ. For twelve years he worked as a carrier without any material disability.

CASE III.—A man, aged forty-one years, who had lived in north-western New South Wales all his life, enlisted in the Australian Imperial Force in June, 1940. Two months later he would notice at the end of the day a pain in the interscapular region, which radiated along the costal margin on the right side. The X-ray film taken on his enlistment was reexamined, and in November he was discharged from the army with the diagnosis of pulmonary tuberculosis. An X-ray examination after his discharge showed a rounded opacity in the posterior part of the left thoracic region at the level of the sixth and seventh ribs. Some destruction of the neck of the left sixth rib was observed. The radiographer suggested a sarcoma of rib as the most likely diagnosis. The patient was admitted to the Royal North Shore Hospital under the care of Dr. M. Susman. At operation a large abscess cavity was found deep to the vertebral end of the sixth rib. The rib showed considerable erosion and gross destruction, and the affected part was removed. Pathological examination revealed a hydatid ectocyst. An interesting feature was that the pain in the right side of the chest was relieved by the operation on the left side. An X-ray picture taken in April, 1941, is said to have shown slight irregularity of the lateral aspects of the fifth, sixth and seventh thoracic vertebræ on the left side.

FIGURE VI.

Case II (tracing of X-ray film). Lateral view shows moderate sclerosis of bodies of sixth, seventh, eighth, ninth and tenth thoracic vertebræ, and complete destruction of intervertebral space between sixth and seventh thoracic vertebræ. Spaces between seventh, eighth and ninth thoracic vertebræ are narrowed. Only slight collapse has occurred of bodies of sixth, seventh, eighth and ninth thoracic vertebræ. In bodies of sixth, seventh, eighth and ninth thoracic vertebræ fairly large cavities are seen. Two small cavities are seen in body of tenth thoracic vertebra. Increased density of anterior surface of bodies of eighth, ninth and tenth thoracic vertebræ is seen with moderate degree of periosteal reaction bridging between seventh and eighth thoracic vertebræ. Cortex of anterior surface of body of seventh thoracic vertebra has been eaten away. In this case chronic infection was superimposed on hydatid infestation.

In 1941 both legs became paralysed. Laminectomy was performed by Dr. Gilbert Phillips and degenerated hydatid material was found to be compressing the spinal cord about the level of the sixth thoracic vertebra. The paralysis disappeared and in August, 1941, the patient commenced work as a labourer. In January, 1942, he enlisted in a garrison battalion, and he was later transferred to the Australian Imperial Force as a signaller. In 1943 pain in the back and paralysis of the legs recurred. After laminectomy on two occasions the paralysis disappeared, and he was discharged again from the army in 1944. Full investigation at the Royal Prince Alfred Hospital in 1944 did not reveal any abnormality. He worked as a salesman without any symptoms until August, 1948, when pain in the back returned and now radiated in girdle fashion at the level of the sixth thoracic vertebra. In October, 1948, he noticed weakness of the left leg. This became more pronounced, and in November the right leg became weak. In December a further laminectomy was performed by Dr. Gilbert Phillips. After operation power returned to the legs and he walked much better. Improvement was only transient, however, and in February, 1949, he developed complete spastic paralysis of the lower limbs. Bladder control was normal, but he lost control of his bowels. Sensation was only slightly interfered with. A further and very extensive laminectomy revealed marked pachymeningitis about the level of the previous operations, that is, from the third to the seventh thoracic vertebra. Further infected hydatid cysts and debris were found. Mild infection developed in the wound. This has been controlled but not eliminated by streptomycin. No material improvement has followed the last operation. X-ray examination on February 10, 1949 (see Figure VII), showed the common picture of hydatid infestation of vertebral bodies, increased density of bodies of the vertebræ, some destruction of bone, no collapse, normal intervertebral spaces and no periostitis. Infection later caused increased destruction of the bodies of the fifth and sixth thoracic vertebræ with collapse.

I was asked to examine this patient because of collapse of vertebræ and girdle pain. Pain was relieved and collapse controlled by continuous traction on skull calipers and the use of a plaster bed.

This case illustrates the extreme chronicity of the disease and the absence of any systemic effects. The involvement of contiguous rib and vertebra is again seen. X-ray examination in 1941 is said to have shown slight irregularity of the lateral surfaces of the fifth, sixth and seventh thoracic vertebræ on the left side. This must have been due to post-operative infection, as X-ray examination in 1944 did not reveal any abnormality of vertebral bodies. Usually when symptoms occur the X-ray signs are advanced, but in this case the first definite evidence of involvement of the vertebral bodies was in February, 1949.

CASE IV.—A man, aged thirty-nine years, was admitted to Sydney Hospital in 1928. He had previously been operated on by Dr. Howard Bullock in 1916 (this case was reported in THE MEDICAL JOURNAL OF AUSTRALIA, Gill and Bullock, 1919). At that time he gave a history of severe pain in the back for two months. He also had paralysis of the lower motor neuron type affecting both lower limbs for one month, and had lost control of his bladder. At the operation in 1916, a hydatid cyst was found in the body of the second lumbar vertebra, with a secondary cyst in the lumbar muscles, and a further cyst in the spinal canal pressing on the *cauda equina*. After the operation, the paralysis of the lower limbs decreased considerably, and he was able to walk with the aid of crutches. He also regained control of his bladder. When he was reexamined in 1919, it was observed that the paresis of the lower limbs was then of the upper motor neuron type. Weakness of his lower limbs eventually increased, and he lost control of his bladder again in 1920. On his readmission to hospital in 1928, examination revealed much wasting of the muscles of both lower limbs with practically no active movement. He had an automatic bladder. There was apparently no sensory disturbance. X-ray examination is said to have shown collapse of the body of the second lumbar vertebra with curvature. At operation on April 13, 1928, a hydatid cyst about the size of an orange

was found to the right of the body of the second lumbar vertebra. After the operation some voluntary control of the bladder returned. The patient subsequently developed an infection in his bladder, and a sinus persisted at the site of operation in the lumbar region. X-ray examination on June 19, 1928, is said to have shown destruction with collapse of the bodies of the twelfth thoracic and first and second lumbar vertebræ. His mental and physical condition deteriorated, and on July 11, 1928, he was transferred to another hospital, where it is believed he died shortly afterwards.

This man lived for thirteen years after the initial successful laminectomy in 1916. X-ray examination twelve years after the first operation is said to have shown collapse of the bodies of the twelfth thoracic and first and second lumbar vertebræ. Dew (1948*f*) states: "It is remarkable how seldom collapse of the vertebral bodies is seen, although it may occur, gradually causing kyphosis in the late stages."

CASE V.—A man, aged fifty years, was admitted to Sydney Hospital under Dr. G. Bell in 1929. He gave a history of severe pain in the right leg and hip for one year and weakness in the right leg for three months prior to admission. He had been unable to walk for one week prior to his admission because both his legs had become weak. He first noticed a swelling in the left upper lumbar region ten days prior to admission. On examination of the patient, a cystic mass was found in the left lumbar region. There were signs of pressure on the right fifth lumbar and first sacral nerves. The right lower limb was much wasted. Hypotonia and weakness of the muscles of both lower limbs were present. At operation on October 16, 1929, a hydatid cyst was found lying beneath the muscles of the back over the laminæ of the vertebræ from the twelfth thoracic to the third lumbar vertebræ. The body and laminæ of the second lumbar vertebra were found to be partly eroded, and a finger could be inserted into the spinal canal. After the operation, paresis of the right leg diminished. X-ray examination on November 23, 1929, is said to have shown some erosion on the left side of the body of the twelfth thoracic vertebra. The patient resumed his usual work on January 25, 1930. The pain in the right hip and weakness of both legs returned after six months, and a further operation was performed on August 27, 1930. He resumed his usual work of farm labourer in May, 1931, and had no symptoms, apart from slight stiffness, in the legs until May, 1932, when he noticed slight pain in the left leg. One further operation was performed in July, 1933, and three additional operations were performed in 1934. These operations were in the nature of further laminectomies, as hydatid cysts gradually spread up the spinal canal in the extra-dural space. At one operation portion of the eighth rib was found to be affected and was excised. Before this patient's death his temperature was high, owing to infection in the wound and to pyelonephritis. Post-mortem examination on May 17, 1935, revealed a cyst, the size of a tennis ball, lying posterior to the bodies of the third and fourth lumbar vertebræ. Small cysts were found in the extra-dural space throughout the specimen, extending from the third thoracic to the fourth lumbar vertebra. This specimen is now in the pathological museum at the Medical School of the University of Sydney.

This case illustrates the extensive spread of cysts in the fatty extra-dural tissue of the spinal cord. It also illustrates that symptoms of pressure from recurrence are usual, but that periods of relief, as in this case, up to two years, may be secured by further operation. Dew (1928*g*) states that, as a rule, muscular weakness, followed by spastic paraplegia with involvement of the spincters and a varying degree of root pain, is the sequence of symptoms when extra-dural hydatid cysts press on the spinal cord. Finally, a true compression myelitis with practically complete motor and sensory loss below the level of the lesion occurs. The development of compression is often extremely slow.

The following case, which is included to illustrate a difficulty in diagnosis, is probably not a case of hydatid disease of bone and has not been considered as such in the statistics.

CASE VI.—A woman, aged twenty-one years, was admitted to the Royal Prince Alfred Hospital under the care of Dr. B. T. Edye in 1935. She gave a history of a swelling in the upper part of the lumbar region, which had been noticed for six months and was gradually increasing in size. At operation, multiple hydatid cysts were found burrowing within the left lumbar sacro-spinalis muscle. Bare bone was palpable behind the sacrum in the region of the left sacro-iliac joint, and also over the left transverse process of the fourth lumbar vertebra. It was noted that there seemed to be an extension into the spinal canal. The patient remained quite well until 1938, when she was readmitted to hospital with the history of backache for sixteen weeks, pain in the right hip and leg for fifteen weeks, and right foot drop for twelve weeks. The pain in the back occurred suddenly and, she stated, had never "let up" since its onset. On examination of the patient, anæsthesia and paresis of the right foot and leg were found. Radiography after lipiodol injection showed a block at the level of the second lumbar vertebra. X-ray examination did not reveal any bony abnormality. The Casoni reaction was obtained. Laminectomy by Dr. R. A. Money on September 20, 1938, revealed multiple hydatid cysts extending extra-durally from the second lumbar vertebra into the sacral canal. After operation slight improvement in movement of the right leg and foot occurred. Anæsthesia was still present over the areas supplied by the fifth lumbar and the first, second and third sacral nerves on the right side. Professor H. R. Dew examined this patient in consultation and considered that the primary hydatid cyst was most likely in a vertebra. I subsequently examined this patient in 1940 because of paresis and anæsthesia of the right foot and have watched her for seven years. I last examined her on August 1, 1947, and found that her condition had not altered. X-ray examination still failed to revel any abnormality of the lumbo-sacral vertebræ, apart from the fact that laminectomy had been performed. A large opacity was, however, seen over the liver area. This was assumed to be an old calcified hydatid cyst of the liver.

Quite logically this patient was believed to be suffering from hydatid disease of bone in 1938, but the subsequent course of events has shown that the condition is more likely a primary hydatid cyst of muscle. I have included this to illustrate a difficulty in diagnosis. The association of multiple hydatid cysts in the muscles of the back and multiple hydatid cysts in the spinal canal strongly suggests that the primary cyst is in a vertebra. However, this patient has remained free from any signs of recurrence for nine years. Dew (1928*h*) states that whenever multiple cysts are discovered near the spine, the bone must be regarded with suspicion and a careful exploration undertaken before the diagnosis of muscle or primary intraspinal cyst is made. Colman in 1899 (quoted by Dew, 1928*i*) collected eight cases from the literature in which no hydatid disease of bone was found, but in which extra-spinal cysts had involved the spinal canal by direct extension. Intra-spinal cysts may also make their way out of the canal, and it is often difficult to determine the primary site of the hydatid disease. In this case, cysts in the muscle were found three years before the onset of symptoms of spinal cord pressure. The cysts in the muscle could still be multiple and yet primary, as a cyst in muscle may be ruptured by muscle action. After rupture, reactive daughter-cyst formation occurs, and a multicystic mass, extending irregularly along the intermuscular spaces, may be produced. When laminectomy was performed, it was found that there were multiple cysts in the extra-dural space of the lower lumbar region. If the primary cyst was in the spinal canal, one would expect a single large cyst, and also expect symptoms of nerve pressure to occur before an extra-spinal extension into the muscles was noted. The age of onset of symptoms suggests that the cysts were not secondary to hydatid disease of bone. If the primary cyst was in bone one would have expected recurrence in nine years. Recurrence may, however, yet take place.

Diagnosis.

The diagnosis of hydatid disease of vertebræ is difficult, but I feel that, as with hydatid disease anywhere, the main thing is to consider the possibility of the disease's being present, and then fewer mistakes will be made. In hydatid disease the general condition is excellent. Symptoms are due to either fracture or subluxation of the affected vertebra or to pressure by extra-osseous extensions. Extra-osseous extensions may be confused with lipomata, or with cold abscess. Extra-osseous extensions are very prone to degenerative change and, as in Case I, the cavity may be filled with yellowish puriform fluid and small daughter-cysts, so that suppuration may be simulated. Bellini (1946) terms these degenerated extensions "ossifluent abscesses of hydatid origin". Dew (1928*j*) states that true suppuration is very rare and usually occurs at a late stage or after operative interference as in Case II.

Symptoms and signs due to spread of hydatid disease into the spinal canal are, of course, indistinguishable from those due to other causes of pressure on the cord. Except in the case of superficial bones, for example, the ribs, the age of onset of symptoms in hydatid disease of bone is late. One should constantly bear in mind the possibility of hydatid disease and undertake Casoni and complement fixation tests. Puncture biopsy can be performed in many cases.

X-ray signs of hydatid disease of vertebræ are not in themselves diagnostic, but when they are considered together with clinical signs, a diagnosis should be possible in most cases. If a patient with hydatid disease of a vertebral body is seen relatively early after the onset of symptoms, X-ray pictures show a much more advanced pathological state than one would anticipate from the duration of symptoms. When, however, hydatid disease primarily affects a pedicle or lamina, the clinical signs and symptoms may be out of proportion to the slight changes seen in X-ray appearance. Microvesicular infiltration of the body of a vertebra destroys the trabecular pattern of the bone. In the vertebræ, when the normal structure of bone is lost, it is unusual to find the rounded cyst-like spaces producing the appearance of a "bunch of grapes" which is seen in hydatid disease of other bones, for example, the ilium. Because of the absence of any tendency to the formation of an osseous cyst-like space, which would weaken the vertebral body, it is not usual to observe any decrease in the height of the body of the affected vertebra. The intervertebral fibro-cartilage is not as a rule severely attacked, and the intervertebral space often remains apparently free of involvement. There is no expansion of the bone. There is no periosteal reaction, except when infection is superadded or as a reaction to injury. The amount of reactive osteitis is variable. In the earlier stages osteolysis predominates, but later, as the result of prolonged irritation, sclerosis may predominate over osteolysis. The invaded vertebra occasionally presents a tendency to eburnation. Massive sequestration is rare, but small sequestra, due to ischæmic necrosis, may give an appearance of spotty recalcification. There is no sharp demarcation between the normal and the diseased areas of bone. When hydatid disease affects a thoracic vertebra the contiguous rib is usually involved. Hydatid infestation of ribs causes a change in the architecture of the affected rib, usually with a predominance of cyst-like rarefaction and moderate osteitis.

In a consideration of the differential diagnosis of hydatid disease of vertebræ the following diseases must be remembered: pyogenic osteomyelitis, tuberculous osteomyelitis, specific infectious spondylitis, secondary carcinoma, hæmangioma, sarcoma, fibrocystic disease, multiple myeloma, solitary bone cyst, syphilitic spondylitis, giant-cell tumour, chondroma and trauma.

In both hydatid infestation and pyogenic osteomyelitis of the spine spread to adjacent vertebræ is common, and in each condition the pedicles may be involved either primarily or secondarily to involvement of the bodies. When pyogenic osteomyelitis affects the body of a vertebra, X-ray examination shows early and marked destruction of the intervertebral fibro-cartilage with consequent narrowing of the intervertebral space. Apart from the very acute type of osteomyelitis, bone destruction is not great and, in consequence, vertebral collapse is not pronounced. Unless the condition of pyogenic spondylitis is recognized early and treated with antibiotics or chemotherapy, reactive new-bone formation and periosteal reaction will be seen as early as six weeks after the onset of symptoms, and after seven or more months bony ankylosis associated with "parrot's beak" formation usually occurs. In pyogenic osteomyelitis clinical and pathological signs of a febrile disease of varying degrees of severity are usually present.

In tuberculous spondylitis early signs are decalcification, narrowing of intervertebral spaces and destruction and collapse of one or more vertebræ. Periosteal new-bone formation occurs late and only in a mild form in uncomplicated Pott's disease. Reactive osteitis is rarely seen in less than one year from the onset of symptoms. Unlike tuberculosis of other joints, tuberculous spondylitis, especially in children, usually leads, after two or more years, to bony fusion of the diseased vertebræ. Cold abscess may resemble extra-osseous hydatid extensions clinically and also on X-ray examination. Calcification in extra-osseous hydatid extensions may simulate the calcareous debris and calcification seen in paravertebral tuberculous abscesses, as in Case I.

The clinical history and the results of agglutination tests, skin tests and blood examination will usually distinguish the different types of infectious spondylitis, for example, typhoid, mumps, Malta fever *et cetera*. The X-ray appearances in specific infectious spondylitis resemble those seen in low-grade pyogenic osteomyelitis. The characteristic finding is narrowing of the involved intervertebral disk, only a portion of the disk being usually involved. Marginal bony proliferation occurs rapidly, and osteophytes typically bridge the affected disk. Solid bony fusion may occur in some cases.

Secondary carcinoma resembles hydatid disease of the vertebræ in that there is a total absence of periosteal reaction and the intervertebral fibrocartilage is resistant to attacks by each disease. Destruction with complete collapse of a vertebra is, however, common in carcinoma. Some types of secondary carcinoma cause a marked reactive new-bone formation.

Hæmangioma produces a honeycomb rarefaction of a vertebral body with collapse and with or without wedging. Reactive osteitis or periostitis is not seen. The involved vertebral body is slightly wider and slightly more compressed supero-inferiorly than normal. The spinous and transverse processes may also be involved, and these have greater dimensions than normal. One or more vertebræ may have these typical appearances. The disks are not involved. The distended vertebral body may compress the cord (Brailsford, 1934). The larger hæmangiomata of the vertebræ produce symptoms in patients over a wide age incidence.

Erosion of vertebræ due to bone sarcoma is most frequently seen in the young. The involved bone is quickly eaten away. The size of the soft-tissue swelling may make this condition resemble hydatid involvement of a lamina or pedicle, in which the extra-osseous tumour may be out of all proportion to the relatively trifling erosion of the bone. One type of sarcoma may be indicated by increased density of the bone. Sarcomata usually show tumour bone formation beyond the normal periphery of the affected bone.

In syphilitic spondylitis (Comroe, 1944) other bones are almost invariably involved. Syphilitic spondylitis has a great predilection for the cervical vertebræ. The vertebræ show bone destruction or new-bone formation or more commonly a combination of each. In most cases the vertebræ become surrounded by spur formation. In a Charcot's spine bizarre destruction of bone with extensive new-bone formation is seen. This new-bone formation may extend beyond the normal limits of a vertebra.

The "bunch of grapes" appearance, commonly seen when hydatid disease affects other bones, is rarely seen in hydatid disease of vertebræ. When, however, this appearance is seen, fibro-cystic disease or multiple myelomata may be simulated. These conditions may usually be differentiated because of multiple bone involvement. The presence of fibro-cystic disease and especially of multiple myelomata is prone to lead to the collapse of one or more vertebral bodies associated with destruction of the intervertebral disks in the latter condition. Ribs affected by myelomatosis are liable to fracture because of the absence of reactive osteitis.

When large cysts occur, as in Case II, the condition must be distinguished from simple bone cyst, chondroma or giant-cell tumour. In hydatid disease expansion of bone does not occur. The cystic space is usually delineated by a thin sclerotic line. Adjacent bone is, however, not normal, and there is no sharp boundary between the normal and the diseased bone. Chondromata lack the extensive and diffuse invasive tendency of hydatid disease, and unlike bone cyst or osteoclastoma are surrounded by sclerosed bone.

Changes in X-ray appearance seen in hydatid disease of vertebræ are not likely to be confused with those of

Kümmell's disease. In the latter condition compression of a single vertebra occurs, usually without narrowing of the intervertebral disks. Usually some periosteal reaction in the form of osteophytes is seen.

Summary.

Six cases of hydatid disease are presented; in five of these the bodies of the vertebræ were primarily affected, in the other case the primary infestation was probably in muscle. The extremely chronic nature of the disease, the absence of systemic effects and the high mortality are illustrated. In Case I, the patient lived five years; in Case II, the patient died, whilst a paraplegic, from misadventure after nineteen years; in Case III, the patient is still alive, although a paraplegic, after nine years; in Case IV, the patient is believed to have died after twelve years (the actual death could not be traced); in Case V, the patient died seven years after the onset of symptoms. Whilst hydatid disease of vertebræ has no pathognomonic radiological signs, certain features, for example, absence of periosteal reaction, minimal collapse, slight involvement only of intervertebral spaces, increased density of bone together with areas of destruction, and when a thoracic vertebra is affected the involvement of contiguous ribs, aid in diagnosis. The most important thing is constantly to bear the disease in mind; fewer mistakes will then be made.

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References.

Bellini, M. A. (1946), "Osteohydatidosis: Its Radiological Features", *Radiology*, Volume XLVII, page 569.

Brailsford, J. F. (1934), "The Radiology of Bones and Joints", First Edition, page 345.

Comroe, Bernard I. (1944), "Arthritis and Allied Conditions", Third Edition, page 665.

Corlette, C. E. (1920), "Hydatid Infestation of Bone, Multilocular Hydatid Disease and Ordinary Hydatid Cysts", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, page 73.

Dew, Harold R. (1928a), "Hydatid Disease", page 370; (1928b), *ibidem*, page 381; (1928c), *ibidem*, page 386; (1928d), ibidem, page 369; (1928e), *ibidem*, page 373; (1928f), *ibidem*, page 386; (1928g), *ibidem*, page 382; (1928h), *ibidem*, page 385; (1928i), *ibidem*, page 385; (1928j), ibidem, page 373.

Gill, James Macdonald, and Bullock, Howard (1919), "Hydatid Disease of the Vertebræ", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, page 336.

Legends to Illustrations.

FIGURE I.—Case I. Note wedging of bodies of second and third lumbar vertebræ, and increased density of first, second and third lumbar vertebræ; the beaking is probably a reaction to subluxation. A few flakes of calcification are visible beyond the outline of the bodies.

FIGURE II.—Case I. Note small flakes of calcification overlying pedicles of second and third lumbar vertebræ. The body of the second lumbar vertebra is compressed and irregular in outline. Joint spaces between bodies are still visible.

FIGURE III.—Case I. (from post-mortem specimen). Note irregular destruction and increased density of bodies of first, second and third lumbar vertebræ, and subluxation between first and second lumbar vertebræ; beaking is probably reaction to subluxation.

FIGURE IV.—Case I (from post-mortem specimen). Note marked destruction of body of second lumbar vertebra with honeycomb appearance and compression. Body of second lumbar vertebra is, however, clearly demarcated from adjacent bodies. Subluxation has occurred between first and second lumbar vertebræ. Note involvement of first lumbar vertebra without any clear line of demarcation between normal and diseased bone; involved bone is denser than normal. Third lumbar vertebra is involved throughout. Increased density of bodies and pedicle of first, second and third lumbar vertebræ is seen with no periosteal reaction and no attempt at ankylosis.

FIGURE VII.—Case III. Note text-book appearance of hydatid infestation uncomplicated by sepsis, namely, increased density of bodies of vertebræ, some destruction of bone, no collapse, normal intervertebral spaces and no periostitis.

PROLAPSE OF THE NUCLEUS PULPOSUS IN THE CERVICAL REGION, ACCOMPANIED BY COMPRESSION OF THE SPINAL CORD, WITH REPORT OF A CASE.

By JAMES ISBISTER,

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THE syndromes produced by prolapse of the *nucleus pulposus* in the cervical region are not generally familiar because descriptions do not appear in the standard textbooks. For this reason the diagnosis in the case to be reported was not made for over six months from the onset of symptoms. The syndrome of cervical root irritation caused by a prolapsed disk has been well described and given some prominence in two articles by Young of Perth (Young, 1945; 1946). In these descriptions brief mention was made of spinal cord compression from a disk, but no account of the clinical features was given. However, the mention of this possibility was the starting point for further investigation and a reassessment of the reported case that led to the correct diagnosis.

When a search of the literature was made it was discovered that the syndrome of spinal cord compression from this cause had been described many times, and numerous cases recorded which were similar to or even identical with the present one. It is nevertheless considered justifiable to draw attention to this syndrome by describing it and recording this case. Undoubtedly similar cases have been missed for lack of familiarity with the clinical picture.

Historical.

Adson (1925) recorded a case of ventral chondroma of the spinal canal causing pressure on the spinal cord at the level of the lower cervical segments. However, the syndrome was first accurately described by Stookey (1928) and this still remains the classical article on the subject. He recorded seven cases of cervical cord compression with unilateral spastic paresis, together with segmental muscle atrophy and fasciculation on the same side, and changes in pain and temperature sensation on the opposite side. The tumours causing the compression were called ventral extradural chondromata. Schmorl (1929) and Andre (1929) first brought forward herniation of the *nucleus pulposus* as a pathological entity. The former, in a series of 3000 autopsies, found herniation of one or more disks into the vertebral canal in 23% of all autopsies, with an increasing incidence with age. Andre described 368 post-mortem examinations, in 56 of which posterior extrusions were found, but in only one case was there compression of the spinal cord. The clinical significance of Schmorl's work and its relation to what had mistakenly been called chondromata, were not realized until Maurie (1933) drew attention to it. This concept was further amplified by Mixter and Barr (1934), who collected 19 cases of prolapsed disk; four were in the cervical region and of these three caused cord compression. Later, Peet and Echols (1934) recorded a case of spinal compression at the level of the third and fourth cervical segments due to a dislocated intervertebral disk. An operation had been performed in 1928, but at the time the true pathological nature of the tumour had not been recognized.

The first review series came from the National Hospital, Queen Square (Hawk, 1936), adding ten cases to make a total of 58 cases in the world literature causing spinal

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ILLUSTRATIONS TO THE ARTICLE BY Dr. LESLIE J. WOODLAND.

Figure I.

Figure II.

Figure III.

Figure IV.

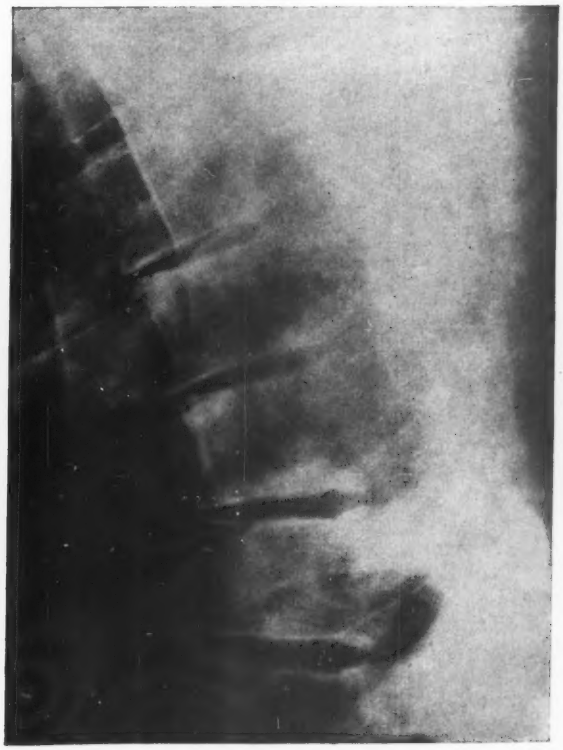

Figure VII.

ILLUSTRATIONS TO THE ARTICLE BY DR. JAMES ISBISTER.

FIGURE II.
Lateral skiagram of the cervical part of the spine. The arrow indicates the osteo-arthritic lipping and narrowed joint space.

FIGURE III.
Oblique skiagram of the cervical part of the spine. The arrow points to the inter-vertebral foramen, in the anterior part of which can be seen the calcification within the neural canal.

ILLUSTRATIONS TO THE ARTICLE BY DR. LAWRENCE W. ALDERMAN.

FIGURE I.
Photomicrograph of section of meninges of midbrain. Low power.

FIGURE II.
Photomicrograph of section of meninges of midbrain. High power.

cord compression; 17 of these occurred in the cervical region. In a series of 100 surgically treated protrusions of the intervertebral disk reported by Love and Walsh (1938) from America, only six had occurred in the cervical region, but all of these had caused compression of the spinal cord.

This review of the literature up to the present decade gives some idea of the growth of knowledge of this subject. It will be noted that in quite a high proportion of reported cases spinal cord compression occurred. However, in the last ten years cervical disk disease has been recognized as a common cause of brachial neuralgia and allied syndromes, and as a result of the present emphasis upon root irritation, spinal cord compression as an effect has received less attention. The best current description of all aspects of cervical disk disease is contained in a book by Bradford and Spurling (1945) entitled "The Intervertebral Disc". The increasing importance of cervical disk disease was further brought to my attention when I attended a discussion at the Royal Society of Medicine opened by Dr. Russell Brain, Mr. Knight and Dr. Bull, and which is reported in the Society's *Proceedings* (Brain, 1948).

Anatomy.

Disk protrusion tends to occur most frequently in the lumbar and cervical regions, because of the lordotic curves, and the relatively great mobility and exposure to trauma, when compared with the thoracic portion of the spine. In the cervical region the spinal cord is anchored by the *ligamenta denticulata* and it occupies a large part of the available space in the neural canal. It is therefore prone to compression by a disk, which in this area is large in relation to the size of the vertebral bodies. In this region the nerves run an approximately horizontal course and are in the neural canal for only a short distance. Consequently, the chance that root irritation will be caused is less than in the lumbar region, and the nerve involved is the one leaving the neural canal at the level of the protruded disk.

The posterior longitudinal ligament is weakest in the middle line, and although expanded at the level of the disk, it does not reach its lateral margin. This accounts for the two commoner sites for pressure by a protruded disk (Figure IA and C), though an intermediate position may result as well. These positions were recognized by Stookey (1928), and the diagrams in Figure I are modified from his paper.

The initial effects of cord compression are vascular, being due to pressure on the venous plexuses. As the flow in these channels is towards the head, œdema of the cord is produced for a short distance below the site of compression. This accounts for the frequently observed fact that the neurological signs are localized one or two segments below the anatomical site of the lesion.

Pathogenesis.

This is a disease predominantly of middle age, and there seems to be some difference of opinion regarding the role played by trauma and degenerative conditions such as osteoarthritis. Horwitz (1940), in 50 autopsies on males between the ages of forty-five and eighty years, found degenerative changes in the disk between the fifth and sixth cervical vertebræ in 76%. This was associated with osteoarthritic changes of the adjacent vertebræ, including exostoses which were present in 70% of cases. This is consistent with the lipping of these two vertebræ frequently seen in radiograms of the cervical spine of patients over the age of forty years. In the series of 100 cases quoted by Love and Walsh (1938) trauma was not an outstanding ætiological factor, whilst Browder and Watson (1945) described a group of 22 cases of cervical disk disease in which it was a possible factor in only five.

On the other hand, some authorities consider trauma to be quite an important cause of prolapse. Kristoff and Odam (1947) have recorded 20 cases of ruptured intervertebral disk in the cervical region, four with cord compression, and they consider injury to be an important causative agent. In certain recorded cases it played a very definite part (Dandy, 1942; Ulmer and Meredith, 1944).

The truth probably is that congenital weakness or degenerative changes predispose to prolapse, which is precipitated by relatively minor trauma. It appears that patients in whom trauma plays an important part are younger than those with osteoarthritis and are more likely to develop root pain than spinal cord compression.

Clinical Features.

The onset of symptoms from a prolapsed cervical disk may be acute, intermittent or insidious. The clinical picture may be due to root pressure, cord compression, or a combination of both (Brain, 1948; Stookey, 1940). The syndrome may be unilateral or bilateral, depending upon the site of the tumour. The onset of cord symptoms is usually insidious and very frequently preceded for some time by root pain (Dandy, 1942; Echols, 1946; Ulmer and Meredith, 1944). In the case described by

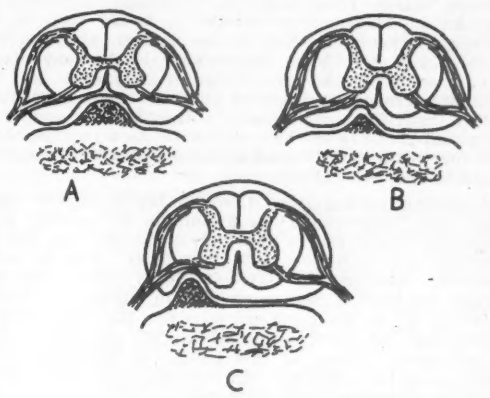

FIGURE 1.

The diagrams illustrate the three positions at which protrusion of the *nucleus pulposus* may take place. A. Central, giving rise to bilateral signs. B. Intermediate, leading to Brown-Séquard syndrome. C. Lateral, associated with root irritation.

Ulmer and Meredith (1944), the patient had brachial neuritis for seven years before the onset of cord compression.

Root compression gives rise to segmental pain in the sensory distribution of the nerve and is well discussed and illustrated by Young (1945, 1946). There is usually diminution of the appropriate tendon reflex, but sensory loss and muscle wasting are minimal. With spinal compression a Brown-Séquard syndrome is produced. The main signs are motor, with segmental lower motor neuron weakness and wasting, and an upper motor neuron effect on the same side below the affected level. The sensory changes are explained by involvement of the lateral spinothalamic tract. This leads to loss of pain and temperature sensation on the whole of the opposite side of the body below the level of the lesion. The dorsal portion of the cord is rarely affected.

As a late sign, involvement of the bladder innervation is important. In a few cases complete transverse myelitis leading to quadriplegia has been reported (Brooke, 1944; Dandy, 1942).

Examination of the Cerebro-Spinal Fluid.

Two changes may be expected in the cerebro-spinal fluid: a raised protein content, and evidence in the Queckenstedt test of partial or complete spinal block. Russell Brain (1948), in a series of nine cases, found a raised protein content in seven and evidence of spinal block in four, with abnormal findings in eight out of the nine cases. Many other writers place little reliance on the cerebro-spinal fluid findings, as they may often be normal (Browder and Watson, 1945).

Radiological Features.

Many papers have been written on the X-ray diagnosis of prolapsed cervical disks. Whilst there is general agreement about the characteristics of the plain X-ray appearances, difference of opinion exists as to the value of opaque myelography, either as a routine procedure or in selected cases. Murphey *et alii* (1946) enumerate the points to be sought in the plain film: (i) scoliosis; (ii) straightening or reversal of the normal cervical curve; (iii) calcification in the posterior part of the joint space; (iv) encroachment of the intervertebral foramen with or without osteophytes; (v) localized arthrosis with narrowing of the joint space. They point out that whilst these changes are not specific they should make one suspicious. Negative findings in the cervical region are of more value than negative findings in the lumbar region.

Both Davies (1945) and Bull (1948) refer to the involvement of the small joints between the lateral margins of the adjacent vertebral bodies that occur only in the lower cervical region (*Uncogelenken*, originally described by Luschka in 1858). This change is better seen in the antero-posterior view than in the lateral, although particularly well seen in the oblique view, as the bony change encroaches upon the intervertebral foramen. At this point pressure may be exerted upon the nerve by changes in this small joint subsequent to disk degeneration, without actual pressure by the disk itself. As a routine, therefore, four plain views should be taken—an antero-posterior, a lateral and two oblique.

Regarding myelography, there is fairly general agreement that "Pantopaque" is preferable to lipiodol, as it gives ample contrast, is relatively non-irritating, has low viscosity for injection and movement, and is easily removed. Epstein and Davidoff (1944) found that with normal subjects it was necessary to invert the patient to 70° to 85° to make iodized oil enter the lower cervical region, and that there is normal slowing and often lack of a continuous column at this point. In patients, partial block could be visualized, but nothing of a lesser nature could be diagnosed with certainty.

Diagnosis.

Cervical disk disease has to be distinguished from a neurofibroma of the dumb-bell type, cervical rib, spinal neoplasm (both intramedullary and extramedullary). Pancoast's syndrome, Hodgkin's disease of the spine, syphilitic pachymeningitis, syringomyelia, *herpes zoster*, arthritis of the shoulder joint and *angina pectoris* (Leigh, 1947). In doubtful cases it is almost always necessary to resort to opaque myelography.

James (1946) considers that all cases that are usually diagnosed as cervical rib or *scalenus anterior* syndrome are in reality cervical disk disease. The hypertrophy of the scalenus muscle is due to spasm, and division leads to relief of pressure on the diseased disk, so accounting for the relief of symptoms.

Prognosis and Treatment.

A pure root lesion may recover with rest, immobilization, or traction. However, there is the risk that later the cord may become compressed, either gradually or suddenly. In the case quoted by Ulmer and Meredith (1944), the patient suffered from "brachial neuritis" for seven years, and then over a period of three weeks developed cord compression, recovery from which was only partial after operation. Two similar cases are recorded by Peron *et alii* (1942). Dandy (1942) quotes a case of pain in the neck and shoulder for six to eight months, followed suddenly by the onset of complete quadriplegia whilst the patient was straining at stool, and death after ten days. He states that this complication has occurred in six out of 300 of his cases. For this reason Bradford and Spurling (1945) in their book advise operation for all cervical disk disease, though this is contrary to the belief of most authorities, who consider it necessary only when there is cord compression or intractable root pain.

Report of a Case.

History.

Mrs. M.C., aged forty-three years, was quite well until the morning of May 31, 1946, when, on waking, she leaned suddenly out of bed to switch on the light and experienced a sudden severe pain in the back of her neck. The pain radiated across her back between the shoulders and was "as if she was being torn apart". After half a minute the pain eased, but thirty minutes later she noticed that her right arm was weak, and that she had an aching pain down the outer aspect of the same arm. She was admitted to Prince Henry Hospital with a diagnosis of poliomyelitis. It was then noted that, as well as having weakness of the right shoulder muscles and biceps group, she had dissociated anæsthesia of the left arm, left side of the trunk and left leg. Lumbar puncture yielded normal findings. The pain in the right arm disappeared after three days. A skiagram of the cervical part of the spine showed osteoarthritic changes affecting the adjacent surfaces of the fifth and sixth vertebræ. Two further lumbar punctures revealed normal cerebro-spinal fluid. After a period of six weeks in hospital she had slight return of power in the right arm. She was discharged from hospital with a diagnosis of hæmatomyelia, and referred to Royal Prince Alfred Hospital for physiotherapy and further supervision.

When examined at this stage (July, 1947) in the medical out-patient department, she complained of weakness of the right shoulder and arm, which was supported in an "aeroplane" splint. She also noticed a sensation of "pins and needles" on the left side of her body and extremities, which was greatest in the left leg below the knee. This symptom was particularly distressing at night, and was associated with a sensation of coldness in the left leg. She complained of no difficulty with walking or micturition.

Physical Examination.

The relevant findings on physical examination of the nervous system were as follows.

1. Motor. There was moderate weakness with muscular wasting of the right deltoid, supraspinatus, infraspinatus, and biceps muscles. There was no muscular fasciculation. There was no weakness or abnormal tone of the lower extremities.

2. Sensory. There was a small area of anæsthesia on the lateral aspect of the right arm, loss of pain and temperature sensation over the left side of the body below the sixth cervical segment, and slightly impaired touch sensation over the same area. Posterior column sensation was normal on both sides.

3. Reflexes. On the right side the triceps and knee jerks were increased when compared with those on the left side. The abdominal reflexes were absent on the right side. The plantar response on the right side was sometimes "extensor", whereas it was always "flexor" on the left side.

Examination of the cervical spine revealed no deformity, limitation of movement or local tenderness. Vertical percussion of the spine was not attempted. There was no Horner's syndrome.

Examination of other systems revealed no abnormality.

No further special investigation was considered necessary at that time, and the provisional diagnosis of hæmatomyelia was not changed. It was resolved to treat the patient expectantly with graduated exercises for the right upper extremity, which was kept in the "aeroplane" splint during the day. Electrical stimulation was also applied at a later stage.

Progress.

The patient was examined at regular intervals, and by February, 1947, there had been some subjective improvement in the power of the right arm, but objectively this was not evident. It was then resolved to review the diagnosis for two reasons: firstly, the patient complained that the paræsthesiæ in the left leg were becoming more marked and unpleasant, and were disturbing her sleep; secondly she insisted that there was something pressing on the "nerves of her neck", and asked for a further surgical opinion. There was no change in the physical findings in the nervous system.

Radiographic examination of the cervical part of the spine again showed osteoarthritic changes in the joint between the fifth and sixth vertebræ, with narrowing of the joint space indicative of disk degeneration (Figure II). An oblique view through the intervertebral foramina on the right side showed an area of calcification in the line of the foramen between the fifth and sixth pedicles (Figure III).

Lumbar puncture revealed normal cerebro-spinal fluid under normal pressure and there was no evidence of obstruc-

tion. The results of Wassermann tests on blood and cerebro-spinal fluid were negative.

Reconsideration of this evidence then led me to the opinion that there was a space-occupying lesion in the spinal canal at the level indicated, and that the pathological condition was probably a prolapsed intervertebral disk. The patient was then (March, 1947) examined by Dr. Morson, who agreed with this possibility, and she was finally admitted to the neurosurgical department of Royal Prince Alfred Hospital under the care of Dr. R. A. Money. The above investigations were repeated with the same results.

Operation.

In July, 1947, laminectomy was performed by Dr. Money at which the laminæ of the fifth and sixth cervical vertebræ were removed. The venous plexus was engorged and bled easily. The right sixth nerve root was coursing upwards and was stretched over a protrusion in the region of the adjacent disk. There were adhesions between the nerve and this protrusion and in the adjoining subarachnoid space. The neighbouring part of the spinal cord appeared to have an increased number of small vessels upon its surface, but was otherwise normal. The "tumour" was removed, and was found to consist mainly of bony material. The histological nature of the excised tissue was reported by Dr. G. E. S. Davies to be *nucleus pulposus* material, mainly osteoid.

Post-Operative Progress.

After operation there was an increase in the weakness of the right arm, but the paræsthesiæ on the left side had disappeared. A further right oblique radiogram of the spine showed that the area of calcification in the intervertebral foramen was now absent.

The patient was discharged from hospital three and a half weeks after operation with her neck supported in a collar, which she was to wear for three months. When she was examined a month after discharge there was slight improvement in the power of the right arm, the sensory improvement on the left side was less, and the extensor plantar response on the right side could not be obtained, though the reflex was not definitely "flexor". The previously disturbing paræsthesiæ were completely absent.

Discussion.

History.

The sudden cord compression was precipitated by relatively minor trauma. As the skiagram of the spine taken when the patient was at the Prince Henry Hospital showed osteoarthritic changes the same as those seen in Figures II and III, it may be assumed that the disk had degenerated previously, and therefore trauma was only a secondary ætiological factor.

The root pain which occurred at the outset lasted only a few days and its subsequent absence accounted for the delay in diagnosis, as one expects this pain before diagnosing a protruded disk. The symptom which really compelled reconsideration of the diagnosis, both by the patient and by the medical attendant, was the distressing paræsthesia of the left leg. This could obviously not be caused by a stationary intramedullary lesion, and therefore I consider it the outstanding feature leading to correct diagnosis.

Physical Examination.

Physical examination revealed a partial Brown-Séquard syndrome, which is typical of these cases. The muscles involved in the right upper extremity, the deltoid, spinati and biceps, are apparently those typically involved in pressure on the sixth nerve, though the anatomical arrangement is obscure.

Cerebro-Spinal Fluid.

The negative findings in the cerebro-spinal fluid are, according to Brain (1948), unusual.

Radiography.

Careful examination of the lateral X-ray view of the spine showed an area of calcification level with the joint space and definitely behind the line of the posterior surfaces of the vertebral bodies. This was later confirmed by examination of the oblique view. In my opinion this appearance suggested a diagnosis that demanded exploration, and therefore myelography was considered superfluous.

Diagnosis.

Other lesions considered in the diagnosis before operation were meningioma, neurofibroma, intramedullary tumour, hæmangioma of the cord and thrombosis of a branch of the anterior spinal artery.

Prognosis.

The original symptoms were probably caused by vascular damage to the cord. With this, and the long duration of compression of the cord, one would not expect much recovery of function after the operation, and this appears to be the case. At least the patient has been cured of the distressing paræsthesiæ, and spared the possibility of a sudden complete spinal compression. It is perhaps surprising that the cord compression did not become greater in the twelve months before operation.

Summary.

1. The syndromes caused by prolapsed intervertebral disk in the cervical region are described, with special reference to compression of the spinal cord.

2. It is emphasized that this is a more common condition than is usually supposed, and a review is made of the numerous reports in the literature. Originally the pathological basis of this syndrome was thought to be a cartilaginous tumour, but about 1933 the importance of the prolapsed disk was recognized.

3. A case is described of sudden partial compression of the spinal cord at the level of the sixth cervical segment by a prolapsed *nucleus pulposus*. The correct diagnosis was not made for six months because of lack of familiarity with the syndrome.

4. Patients presenting with this condition have a sudden or gradual onset of a Brown-Séquard syndrome with or without preceding cervical root pain. Trauma is not an important ætiological factor. The diagnosis is very much facilitated by radiological examination of the cervical part of the spine in the antero-posterior, lateral and two oblique views. Less assistance is gained from cerebro-spinal fluid examination, though a high protein content and evidence of partial or complete block may be obtained. Opaque myelography is not often necessary.

5. In this syndrome operation is definitely indicated when there is cord compression, though much improvement in the cord damage cannot be expected. Without operation there is a serious risk of gradual or sudden complete cord compression.

Acknowledgements.

I wish to thank Professor C. G. Lambie, in whose department I was working when this case was under my notice, for his help with this paper, and Dr. R. A. Money, who performed the operation, for his permission to report this case. I am indebted to Mr. Woodward-Smith for the reproductions of the skiagrams.

References.

Adson, A. W. (1925), "Diagnosis and Treatment of Spinal Tumours", *Northwest Medicine*, Volume XXIV, page 309.

Andre, R. (1929), *"Ueber Knorpelknötchen am hinteren Ende der Wirbelbandscheiben in Bereich des spinalkanals", Beiträge zur pathologischen Anatomie und zur allgemeinen Pathologie*, Volume LXXXII, page 464.

Bradford, F. K., and Spurling, R. G. (1945), "The Intervertebral Disc", Second Edition.

Brain, W. R. (1948), "Discussion on Rupture of the Intervertebral Disc in the Cervical Region", *Proceedings of the Royal Society of Medicine*, Volume XLI, page 509.

Brooke, W. S. (1944), "Complete Transverse Cervical Myelitis Caused by Traumatic Herniation of an Ossified Nucleus Pulposus", *The Journal of the American Medical Association*, Volume CXXV, page 117.

Browder, J., and Watson, R. (1945), "Lesions of the Cervical Intervertebral Disc: Clinico-Pathological Study of Twenty-two Cases", *New York State Journal of Medicine*, Volume XLV, page 730.

Bull, J. W. D. (1948), "Discussion on Rupture of the Intervertebral Disc in the Cervical Region", *Proceedings of the Royal Society of Medicine*, Volume XLI, page 513.

Dandy, W. E. (1942), "Serious Complications of Ruptured Intervertebral Discs", *The Journal of the American Medical Association*, Volume CXIX, page 474.

Davies, H. (1945), "Lateral Prolapse of the Cervical Intervertebral Disc", *The British Journal of Radiology*, Volume XVIII, page 1.
Echols, D. H. (1946), "Radicular Pain Due to Ruptured Cervical Disc", *The Southern Surgeon*, Volume XII, page 205.
Epstein, B. S., and Davidoff, L. M. (1944), "Iodised Oil Myelography of the Cervical Spine", *The American Journal of Roentgenology and Radium Therapy*, Volume LII, page 253.
Hawk, W. A. (1936), "Cervical Compression Caused by Echondrosis of the Intervertebral Fibrocartilage; with a Review of the Recent Literature", *Brain*, Volume LIX, page 204.
Horwitz, T. (1940), "Degenerative Lesions in the Cervical Portion of the Spine", *Archives of Internal Medicine*, Volume LXV, page 1178.
James, E. S. (1946), "Protruded Cervical Intervertebral Discs—A Cause of Brachial Neuritis", *The Canadian Medical Association Journal*, Volume LV, page 139.
Krisroff, F. V., and Odam, G. L. (1947), "Ruptured Intervertebral Disc in the Cervical Region; A Report of Twenty Cases", *Archives of Surgery*, Volume LIV, page 287.
Leigh, A. D. (1947), "The Prolapsed Intervertebral Disc", *The Post-Graduate Medical Journal*, Volume XXIII, page 141.
Love, J. G., and Walsh, M. N. (1938), "Protruded Intervertebral Discs: Report of One Hundred Cases in Which Operation was Performed", *The Journal of the American Medical Association*, Volume CXI, page 396.
Maurie, G. (1933), "*Le disque intervertébral: physiologie, pathologie et indications thérapeutiques*".
Mixter, W. J., and Barr, J. S. (1934), "Rupture of the Intervertebral Disc with Involvement of the Spinal Cord", *The New England Journal of Medicine*, Volume CCXI, page 210.
Murphey, F., Pascucci, L. M., Meade, W. H., and van Zwaluwenburg, B. R. (1946), "Myelography in Patients with Ruptured Cervical Intervertebral Discs", *The American Journal of Roentgenology and Radium Therapy*, Volume LVI, page 27.
Peet, M. M., and Echols, D. H. (1934), "Herniation of the Nucleus Pulposus; A Cause of Compression of the Spinal Cord", *Archives of Neurology and Psychiatry*, Volume XXXII, page 924.
Peron, N., Lereboullet, J., Guillaume, J., and Dumas, C. R. (1942), "*Hernie discale cervicale déterminant dans deux cas un tableau de compression médullaire avec syndrome de Brown-Séquard; opération guérison*", *Revue Neurologique*, Volume LXXIV, page 306.
Schmorl, G. (1929), "*Über Knorpelknoten an der Hinterflache der Wirbelbandscheiben*", *Fortschritte auf dem Gebiete der Röntgenstrahlen*, Volume XL, page 629.
Stookey, B. (1928), "Compression of the Spinal Cord due to Ventral Extradural Cervical Chondromas", *Archives of Neurology and Psychiatry*, Volume XX, page 275; (1940), "Compression of the Spinal Cord and Nerve Roots by Herniation of the Nucleus Pulposus in the Cervical Region", *Archives of Surgery*, Volume XL, page 417.
Ulmer, J. L., and Meredith, J. M. (1944), "The Syndrome of Cervical Root Compression and Brachial Neuritis following Lateral Herniation of the Intervertebral Disc, with Comment on Central Midline Protrusion", *Virginia Medical Monthly*, Volume LXXI, page 119.
Young, J. H. (1945), "Intervertebral Disk Disease: New Syndromes and New Concepts", THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, page 234; (1946), "Cervical and Thoracic Intervertebral Disk Disease", *Ibidem*, Volume II, page 833.

Reports of Cases.

REPORT ON A CASE OF CEREBRO-SPINAL MENINGITIS DUE TO CRYPTOCOCCUS HISTOLYTICA (TORULA HOMINIS).

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THE purpose of this report is to place on record the clinical course and pathology of cerebro-spinal torulosis, as seen in a half-caste adult male aboriginal at Alice Spings, Central Australia. Mention will also be made of another case of torulosis in a full-blooded aboriginal at Tennant Creek, 300 miles north of Alice Springs, which occurred some sixteen months previously. In my opinion, the excessive rarity of this condition renders the reporting of these cases worth while, for as far as I can ascertain, there have been only about twelve cases described in Australia, and about 100 cases mentioned in general medical literature.

Clinical Record.

Mr. D., the half-caste patient, aged twenty-six years, was first seen in the out-patient department of the Tennant Creek Hospital, where he complained of headache and constipation, and was given "A.P.C." for the former and a purgative for the latter. He was then *en route* for Alice Springs, and on arrival here on October 21, 1947, he reported again to the out-patient department with the same symptoms. Examination revealed no abnormality in the central nervous system, the sinuses were normal, the blood pressure was 120 millimetres of mercury (systolic) and 70 millimetres (diastolic), and the urine was normal. Similar treatment to that given at Tennant Creek was prescribed, and he was not seen again for ten days; then he reported at the out-patient department with an exacerbation of the headache and a pain in the neck.

A dramatic deterioration was observed in the patient's condition. He had lost sixteen pounds in weight in the ten days, he had an anxious expression and he was vomiting profusely and often. The clinical examination revealed no abnormality except pronounced bradycardia and a peculiar appearance of the left optic disk, which was hazy in outline and presented the appearance of early papilloedema. There was a patch of chorioiditis in the superior nasal quadrant of the same eye.

Spinal puncture was performed, revealing cerebro-spinal fluid at very high pressure (it shot out of the spinal puncture needle) with increased protein content and 50 cells per cubic millimetre. X-ray pictures of the skull were taken, and the lateral view was interesting in that a marked prominence was observed in the occipital area, suggesting the possible presence of a tumour in this region, a diagnosis which up till now had been foremost in the diagnostic field. The report on the film, however, was returned as "batrocephaly within normal limits" (batrachocephaly, or frog head).

At this stage, the patient began to record a considerable pyrexia with an evening peak, and the diagnosis rested between influenzal and tuberculous meningitis. Accordingly *Hæmophilus influenzæ* antiserum 30 millilitres, penicillin 25,000 units three-hourly, and sulphadiazine were administered, and X-ray examination of the chest was made. The latter showed that the heart and lung fields were radiologically normal. The probability of tuberculosis seemed reasonable as enquiries amongst his relatives elicited the information that he had been under treatment for severe lung trouble during the military occupation of this area. The eventual diagnosis of torulosis is interesting in this respect, as the possibility of a primary pulmonary infection with cryptococcus must be considered. Fortunately, culture of the cerebro-spinal fluid with Dorset's egg medium, Löwenstein's medium, and Petrangnani's medium later on yielded a contaminated culture of the cryptococcus.

Throughout the course of the disease, the patient's main symptom was excruciating headache, which at times made him scream with pain; at least half a grain of morphine was required to relieve it. Associated with a blurred optic disk and bradycardia, it was assumed that the headache was due to increased intracranial pressure, and frequent spinal punctures were performed, but with only temporary relief from the decompression. Each specimen was examined and the findings in each varied very little from the original specimen. Enemata of 25% magnesium sulphate solution were also employed, but they were expelled as soon as given.

On November 17, 1947, while the central nervous system was being examined during ward rounds, it was noted that both knee jerks were absent. The plantar reflexes were both normal and the ankle jerks were present. At this stage, the diagnosis was considered to be diffuse idiopathic encephalomyelitis, and so closely did the course of the disease resemble a diffusing myelitis that I persisted in this diagnosis till the specimens obtained at post-mortem examination were reported on from Adelaide.

The next day his ankle jerks were absent, he complained of violent pains in the thighs and abdomen, and he was tender on pressure in both iliac fossæ. Some alarm was felt in case an abdominal condition had been susperimposed, but the abdominal wall was lax, the bradycardia persisted, and two days afterwards the pains were generalized. From his description, they were apparently due to involvement of the posterior spinal nerve roots.

He then complained of numbness of the forehead, and very soon patches of anæsthesia and paræsthesia appeared elsewhere. His vision became poor, photophobia and diplopia developed, the pupils became fixed, and nystagmus was observed. At this stage, the left optic disk was frankly papillœdematous, the right was slightly hazy in outline, the upper limb reflexes were absent, and he developed a right abducens paralysis. Emaciation was extreme and dysphagia had made itself apparent, so that it was difficult to continue administering fluids by mouth. At the same time, sphincter control disappeared, and in the usual direct proportion, bedsores appeared. The patient became odd mentally, and kept repeating that he was as fit as a fiddle, a statement singularly in contrast with his appearance.

Unfortunately, he had been a strong lad and his resistance was amazing. Though leading practically a vegetative existence, so extremely emaciated that he was literally only "skin and bone", ataxic, continually incontinent, demented, and yet still in severe pain, he lived for about ten days on morphine, receiving half to one grain when necessary—a strong argument for euthanasia.

Autopsy Findings.

The post-mortem findings were as follows.

1. Except for areas of perihepatits, the heart, lungs, liver, kidneys *et cetera* were all normal on microscopic examination.
2. The brain substance seemed to be very soft and the *pia mater* was surrounded by a thick slimy layer of mucoid cerebro-spinal fluid.
3. When section was made of the spinal cord at intervals the nervous substance oozed through the incisions, reminding one irresistibly of myelitis.

Histological Examination.

Dr. M. Fowler reported that examination of the sections showed a torula meningitis. In none of the sections made was there any evidence of invasion of the brain substance. Tissue from the cord, cerebellum and temporal lobe was submitted, and each showed the typical lesions in the meninges. The intensity of the infection was greatest over the hippocampus.

The organism occurred singly or in small collections, and in a few places in large numbers, and appeared as a round grey body or as a clear body with a doubly refractile wall. It was invariably surrounded by a clear zone, into which there sometimes radiated spine-like processes from the organism.

The reactive areas in the meninges contained lymphocytes, plasma cells, a few endothelioid cells, and an occasional giant cell. Necrosis of the exudate was evident in some places. The blood vessels, medium and small, had surrounding collars of round cells, mainly lymphocytes (see Figures I and II).

Cultural Characteristics of the Organism.

As has been noted, culture of the spinal fluid was made on Dorset's egg medium, Löwenstein's medium and Petragnani's medium. When the diagnosis based on the post-mortem sections was received, the cultures were reexamined and typical, strongly Gram-positive torula cells were seen in formalin-fixed smears from the cultures on Petragnani's medium. In some cases, these were surrounded by daughter cells, and they were always provided with a definite capsule or mucinous envelope. Two examples of budding from the torula cells were discovered. Subcultures were made on to Sabouraud's medium, and a thick, moist, creamy growth occurred at 37° C. in forty-eight hours. This consisted of similar yeast cells to those seen in the original culture.

Summary of Laboratory Findings.

The total of white cells in the blood varied from 12,000 per cubic millimetre to 25,000 per cubic millimetre, the greater proportion of the increase in all cases being due to granulocytes. The results of blood culture and Weil-Felix tests were negative, while the result of the Widal test was positive in the lower dilutions, a finding apparently due to military "T.A.B." inoculation. Repeated examination of the cerebro-spinal fluid revealed up to 70 cells per cubic millimetre, an increased globulin content, and the presence of a protein pellicle after the fluid had been standing; the colloidal gold estimation revealed a meningitic curve. The hæmoglobin value on November 17, 1947, was 75% (Sahli).

The Tennant Creek Case.

Mention has already been made of a previous case which occurred at Tennant Creek in 1946. I am also informed that there have been several cases of pulmonary torulosis in Darwin since the Alice Springs case. The patient in the Tennant Creek case was a full-blooded aboriginal woman, aged about twenty years, who had been admitted to hospital with a febrile illness extending over a month; she had pain on the left side of the neck, limitation of flexion of the neck, weakness of both legs, headache and ensuing blindness. Both defæcation and micturition remained unimpaired till the last day. No sign of trauma was found at initial examination or autopsy. Unfortunately further details are unavailable, except the fact that on examination of the white matter (one specimen in the parietal lobe and the other in the mid-brain), cortical lesions of torulosis were discovered.

Commentary.

It would be useless to attempt to compare the two cases, as so little is known of the Tennant Creek case, but it is noted that the meningitic type was accompanied by an intermittent pyrexia, while the cortical type ran an afebrile course. It is also observed that defæcation and micturition were unimpaired till the last day in the Tennant Creek case, whereas in the last fortnight the Alice Springs patient presented the appearance of having extensive acute myelitis, with sphincter loss a marked feature.

Ætiology.

A guess could be hazarded that different varieties of the cryptococcus are responsible for different types of torulosis. In some cases only localized lesions, for example, of the tibia, are observed and these are sometimes curable. In the Alice Springs case, the meninges came in for special attention from the yeast; in the Tennant Creek case the cortex was attacked, and in other cases there has been a generalized systemic invasion of the lungs, liver, spleen, kidneys *et cetera*. Conditions involving the central nervous system and generalized conditions of torulosis are invariably fatal.

Mode of Entry.

No evidence is available as to the mode of entry in either case. The only suggestive fact is the statement by one of the relatives of the Alice Springs patient, that the patient had an attack of "severe chest trouble" in the army days. However, I could find no subpleural lesions, or any involvement of the lungs at all for that matter in this case. There were several patches of perihepatitis, but unfortunately sections of these were not examined.

One interesting point is that both patients contracted the disease in Tennant Creek, so that this town certainly has had more than its share of the reported cases in Australia.

Clinical Course.

Fever is said usually to be absent, but the Alice Springs patient had quite a high intermittent pyrexia, with peaks up to 103° F., extending over the last four weeks of his illness. The Tennant Creek case ran an afebrile course.

The presenting symptom, and always the most obvious one, was an excruciating headache. Bradycardia was an early sign, the patient's pulse rate even when he was pyrexial being as low as 44 per minute. As the cerebro-spinal fluid was under very high pressure and papillœdema was present early in the piece, it is reasonable to assume that increased intracranial pressure plays a large part in the symptomatology, so that, firstly, intracranial tumour, and, secondly, tuberculous meningitis are simulated.

The first alteration in the reflexes came with the loss of the knee jerks on both sides, but as the disease progressed

paraplegia developed and the process rapidly involved the cranial centres as well, with resulting nystagmus, strabismus, diplopia, facial anæsthesia and asynergia. As has been noted, girdle pains were so severe as to indicate an abdominal catastrophe, and indicated involvement of the posterior nerve root areas. The patient died two months after the onset of symptoms.

Investigations.

The laboratory pathological findings fitted in with the usual picture of the disease. There was leucocytosis, with an increase in the proportion of granulocytes. There was no eosinophilia. Later in the course of the illness mild anæmia developed. Culture of blood did not produce a growth of any of the cryptococci in plain broth. Culture of the cerebro-spinal fluid on Petragnani's and later on Sabouraud's medium yielded a growth of the cryptococcus.

The cerebro-spinal fluid itself was colourless, with an increase in globulin content, a protein pellicle on standing, and a cell content of up to 70 cells per cubic millimetre. X-ray examination of the chest was made, but the heart and lung fields were normal. The X-ray appearance of the skull has already been mentioned.

Treatment.

I feel that I am unable to amend, from my experience with this case, the gloomy outlook that all cases of cerebro-spinal or pulmonary torulosis are invariably fatal. In this case the patient received 2,275,000 units of aqueous penicillin over twelve days, and 65 grammes of sulphadiazine. Iodides were tried without success, and when myelitis seemed a likely diagnosis, massive vitamin *B* therapy was given. With the possibility of tuberculous meningitis in mind, an attempt was made to obtain streptomycin, but none was available in Adelaide at that time; and even so, I can find no record of yeast's being sensitive to either penicillin or streptomycin in the medical literature at my disposal.

Summary.

1. A case of meningitis due to *Torula histolytica* is described.

2. A second case of cortical torulosis is mentioned.

3. No reason was found to suppose that torulosis is susceptible to sulphadiazine or penicillin.

Acknowledgements.

I wish to thank Dr. M. Fowler and Professor J. B. Cleland for their reports on the sections, Dr. V. R. Webster for his assistance with the Tennant Creek case, and the Director-General of Health for his permission to publish this report.

Reviews.

PHARMACOLOGY IN PRACTICE.

"THE PHARMACOLOGIC PRINCIPLES OF MEDICAL PRACTICE", by John C. Krantz, junior, and C. Jelleff Carr, presents a full range of currently used drugs with free discussion as to the mechanisms of their actions and to their clinical applications.[1]

In selecting the title, the authors define their approach succinctly by defining "principles" and "practice" respectively as "what to do and why", and "how to do it and when". A study of the treatise indicates that they have amply fulfilled the promise inherent in this approach.

The arrangement of the subject matter is in several parts, each dealing with the responses of individual systems to appropriate drugs. Each part is introduced by clearly enunciated physiological and biochemical data on which relevant pharmacological applications depend. While this classification has many advantages, it often involves much cross reference if one desires to review the complete pharmacology of an individual drug.

The matter is set out in an easy fluent style, with prefatory historical references where indicated, and freely illustrated with finely produced photographs of outstanding investigators in each field under review.

While dismissing with brief comment many older drugs still in current use, the authors rightly give much space and detail to more modern drugs. Particularly interesting chapters are devoted to such themes as the autonomic nervous system and the many synthetic cholinergic and adrenergic drugs, the response of water balance to pharmacologic agents, iodine metabolism and antithyreoid drugs, the response to histamine and the antihistamines, and the relative value of antimalarials—to mention but a few.

The wider scope of the work may be estimated by its reference to such subjects as the cytotoxic action of the nitrogen mustards, the nature and use of radio-active phosphorus, the methylating and the lipotropic functions of choline, and the "enhancement" factor in penicillins of commercial purity. Pharmacological evidence both for and against the use of many individual modern drugs is well presented and witnesses both for their arraignment and their defence are quoted freely and supported by a limited, but useful, bibliography. The least convincing portions are those very minor ones devoted to the prescriptions and to occasional pharmaceutical procedures. The authors might with advantage follow their reiterated advice—"confer frequently with your pharmacist".

Despite this almost negligible criticism it is a satisfying scholarly work, admirably produced and affording both pleasure and profit in its study. It may be recommended as meriting its place among the books of daily reference.

[1] "The Pharmacologic Principles of Medical Practice: A Textbook on Pharmacology and Therapeutics for Medical Students, Physicians, and the Members of the Professions Allied to Medicine", by John C. Krantz, junior, and C. Jelleff Carr; 1949. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" x 5", pp. 900, with 94 illustrations, some of them coloured. Price: 75s.

ROLE OF HEREDITY AND HORMONES IN SEX.

IN cheap brochures constituting a series emanating from an enterprising publishing house the aim is generally to present in each volume a synopsis of some scientific problem or current question of the day. Too often the author chosen to deal with some aspect of his specialty uses this medium to air views peculiarly his own, but in *"Le sexe"* by Vera Dantchakoff the author has embodied in a book of unpretentious size the results of an immense amount of research conducted by herself on a wide range of animals and dealing with the important and manifold problems of the determination of sex and the interrelations between sex and heredity.[1] The experiments must have taken years to carry out, and many of them were beset with considerable manipulative difficulties. The animals employed were frogs, lizards, birds (here Leghorn fowls proved to be best suited to the purpose), guinea-pigs, rats, mice and monkeys. One expects in a French publication pellucid clarity of expression, but we must confess to occasional perplexity as to the meaning and logical consistence arising probably from the concentration which the small size of the book necessitated. Amongst the important conclusions are the following. The sex of the offspring is determined at the union of ovum and spermatozoon. In the mammal it is the male parent which imposes maleness or femaleness on the embryo as 50% of the spermatozoa carry male determinants and 50% female. Hence artificial modification of the distribution of the sexes would need human intervention before fertilization; eliminate from the sire the determinants of one sex and then the other will alone appear. There are difficulties such as true hermaphroditism, and even more so in those animals whose sex can change in the adult, and it is here that the author's explanations are difficult to follow. When the sex of the embryo has been decided, then the appropriate hormone, folliculin or testosterone, takes charge. There are most interesting accounts of the folliculinized embryo males developing female characteristics and testosteronized embryo females displaying male features. It is noteworthy that after an injection of folliculin a chick rooster will show all the qualities of a pullet, but after a time maleness triumphs and the comb, wattles and spurs become exceptionally large. The book ends with a curious disquisition on the philosophy of sex as meeting the universal love of life and fear of death. The final sentence is a denunciation of those Russian biologists who subordinate scientific truth to political theory. The book must be taken seriously by all students of genetic research. There is a bibliography but no index.

[1] *"Le sexe: rôle de l'hérédité et des hormones dans sa réalisation"*, by Vera Dantchakoff; 1949. Paris: Presses universitaires de France. 7¼" x 4¾", pp. 218, with many illustrations. Price: fr. 480.

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THE SINS OF MEDICINE.

In a lecture delivered at London to the University College Hospital Medical Society on March 17, 1948, Richard Asher, physician of the Central Middlesex Hospital, dealt with what he called the seven sins of medicine.[1] He was careful to say at the outset that there were an unlimited number of medical sins, but that he intended to deal with the following seven: obscurity, cruelty, bad manners, over-specialization, love of the rare, stupidity and sloth. Asher no doubt chose sins to the number of seven because of the traditional seven deadly sins—anger, avarice, lust, pride, envy, gluttony and sloth. We may almost certainly disregard the idea that in his discourse he had time to deal with only seven. There is something subtle about the number seven; it has been called the perfect number. It has been described as a mystical and symbolical number in the Bible as well as among the chief nations of antiquity. Among the reasons offered for the preference for seven are the suggestions that three stands for the spiritual (the Trinity) and four for the material (the four elements), the combination representing the supernatural sphere. The main reason is probably bound up with the observation of the seven planets and the phases of the moon, changing every seventh day. Anyhow, we read in the Book of Proverbs that Wisdom built her house with seven pillars; there seems to be no reason therefore why Folly should not bring a man of medicine, if he is malleable, to his undoing with seven sins.

What, it will be asked, is sin? Sin may be defined as transgression against a moral code. The code is usually that of religion, but in modern times the idea is extended to include ethics of society and of its creations. Thus it is not only permissible, but logical, to refer to sins against medicine—its tradition, its content and its standard. Let us look at Asher's seven sins in turn. He describes obscurity in writing and in lecturing and holds that it is bad, "not only because it is difficult to understand, but also because it is confused with profundity, just as a shallow muddy pool may look deep". Obscurity is sometimes used in the hope that it will be confused with erudition. People who do this refrain from the use of a short word, if a longer can be found. Asher remarks that if a patient says "If I go half-way up a hill I feel I'm done for", these very words should appear in the notes and not, "The patient complains that during ambulation up a moderate incline he suffers a feeling of impending dissolution". Obscurity and slovenliness of language often go hand in hand because an attempt is not made to convey an exact meaning. The surgeon was not really erudite who wrote that "in malignant disease of the breast it may safely be predicted that death will occur unless some other fatality intervenes". Cruelty is named by Asher as probably the most important and prevalent sin in the list chosen by him. Let us agree at once, for to be cruel is to be false to every true conception of medicine. Asher names mental and physical cruelty. The former is common and arises in three ways: (*a*) by saying too much, (*b*) by saying too little, (c) by the patient being forgotten. There should be no need to deal with these in detail. We all know how cruelty may be inflicted by saying too much, but we are apt to forget that the patient is generally an intelligent person and may be hurt equally when we say too little. In the one case we heap onto a patient a burden of anxiety, in the other we leave a gap through which the patient may build up for himself a burden of fear and anxiety. Both types of burden may become intolerable. The third type of mental cruelty, caused when the patient is forgotten, has to do chiefly with clinical teaching when the patient is treated almost as if he was unconscious. But remarks to relatives at the bedside and in the home of the patient, when it is thought that the patient does not hear or heed, may be just as harmful as when they are made in a teaching hospital. Physical cruelty is caused by over-investigation. Asher has pertinent remarks to make about clinicians who "hunt the primary" in a patient dying of cancer in the way that they might play "hunt the slipper". He has something to say about the use of cardiac catheters, arterial punctures and so on in a patient dying of heart failure. He also deprecates the administration of a test meal to a patient with pernicious anæmia whose hæmoglobin is "in the twenties". "Do not wheel elaborate trolleys to a patient's bedside and start sticking needles in him until you have given him a word of reassurance and explanation." The third sin is bad manners. They are dealt with under three headings—towards patients, towards nurses and towards medical staff. The fourth sin is over-specialization, whose worst feature is that "it makes doctors feel that they are doing wrong to deal with even the simplest case if it lies within the protected area of somebody else's specialty". Then comes "love of the rare". This sin is chiefly found among students and is responsible for many failures at examinations. The sixth sin is common stupidity. Of course, if a man is congenitally stupid he should not study medicine. Asher refers to the commonest type of stupidity which he calls therapeutic automatism. In this a rigid code of treatment is advised in all circumstances. "One must cut one's therapeutic coat according to the mental and economic cloth of one's patient." To order an elaborate and expensive diet for a patient who has to work hard and perhaps live alone is the height of stupidity. The last sin is sloth, which may be physical or mental. Physical sloth may cause the omission of certain important clinical

[1] *The Lancet*, August 27, 1949.

examinations and may lead to lapses in aseptic ritual. Mental sloth is commoner. It may lead to a too ready acceptance of a patient's statements and even of his diagnosis of his own complaint. It may lead to too ready an acceptance of the written word. Of course, personal sloth of the doctor should be mentioned—physical sloth in which he neglects to take exercise for his body and failure to exercise his mind in pursuits outside clinical medicine. Sloth of this kind, though not mentioned by Asher, is most reprehensible.

Asher's "seven sins" have been mentioned. They have been well chosen. It would be safe to say that a practitioner who managed to avoid them all would avoid most of the other sins which any reader could add to Asher's list. Three come to mind at once—greed, conceit and intellectual dishonesty. It will be said that the avoidance of these sins depends largely on teaching in university and hospital. This is, of course, true, but a good deal also depends on the calibre of the person who is being taught, whether the soil in which the seed of teaching will grow is well prepared and contains the necessary components which will stimulate and perfect growth. The things which undergraduate students in medicine should learn were well stated by Professor F. A. E. Crew in an address to new medical graduates at Edinburgh on July 20, 1949. (The address is printed in the same journal as Asher's article.) He asked:

> Have you, as a result of our intervention in your affairs, become or remained capable of critically observing, of skillfully experimenting, of discriminating between the true and the false? Have we made you more capable than before of entertaining a new idea, a friend, and, especially, yourself? Have we by our advocacy and example taught you to loathe intolerance, intellectual dishonesty, and inaccuracy? Have we in our behaviour persuaded you to avoid malice, bitterness, and envy and to cultivate a constructive discontent concerning not only things in general, but also, and especially, yourself? Have we played a part in the fashioning of your secret ambition? Have we taught you to seek, not wealth, not security, but the understanding heart?

Life spent in the practice of medicine is just like life spent in any other avenue. We may begin with a full and adequate equipment, but we soon discover that the road is not easy. To speak in the language of the theologians—it is easy to fall into sin, the sins of which Asher has written and those others which each of us can add to his list. Only by constant watchfulness and by refreshment from sources well known to us can we sustain ourselves and add lustre to our calling.

Current Comment.

THE FATE OF SUBSTANCES INJECTED INTRATHECALLY.

IT is probably not ungenerous to say that many of those who inject substances into the spinal theca give little more thought to where those substances go than they give to the hibernation of *Musca domestica*. Of the fate of such radio-opaque substances as lipiodol we are left in little doubt; they remain, at least for some time, as radiologically visible foreign bodies, *aides-mémoire* to the principle that they may be used only if the information to be obtained by their injection outweighs in importance the harm that may be done by their continued presence in the theca. Their removal after their purpose has been served is advocated, but to effect this completely is not always easy. That iodized oils are at least potentially harmful has been often pointed out, not least in a special report of the Council on Pharmacy and Chemistry of the American Medical Association as far back as 1932.[1] The Council found then that careful surgeons were inclined to look on subarachnoid injections of iodized oils as "a dangerous last resort". At the other end of the scale from these substances are those which appear in the blood-stream soon after their intrathecal injection. Of therapeutic substances which undergo absorption into the blood-stream G. Wilson *et alii*[2] suggest that probably any benefit from their intrathecal injection occurs only after this absorption has taken place; this forms part of an argument urging the greatest caution in the use of intrathecal injections, to which detailed reference has already been made in these columns (see THE MEDICAL JOURNAL OF AUSTRALIA, November 19, 1949). Just how much substances in the blood-stream, whether of intrathecal or other origin, can enter the cerebro-spinal fluid is considerably disputed and cannot be gone into here; attention may, however, be drawn to recent work by J. W. T. Redfearn, A. Elithorn, K. Till and F. A. Ibbott[3] in which they have found that, contrary to the statements of a number of previous investigators, the normal barrier between blood and cerebro-spinal fluid is permeable to penicillin, and that this permeability is increased by organic disease of the central nervous system, such as neurosyphilis.

Of quite different significance from the fate of therapeutic substances is that of anæsthetic agents introduced into the spinal theca. A. Goldberg, H. Koster and R. Warshaw[4] investigated the fall in concentration of procaine after its intrathecal injection, and concluded that absorption into the blood-stream occurred, being followed by rapid detoxication (probably achieved by enzymatic action), so that at no time was there any appreciable trace of procaine itself in the blood; the products of detoxication left the blood-stream rapidly until equilibrium was approached between the blood and other tissues, nearly all the injected procaine (90% on the average) being finally excreted in the urine, but only in the form of products of detoxication. In a discussion on spinal anæsthesia in 1929, R. B. Stout[5] accepted the idea of "fixation" of the anæsthetic agent by the spinal nerve tissue as an important factor in limiting the upward spread of anæsthesia; but Goldberg *et alii* point out that while their own findings do not elucidate the mechanism of the production of anæsthesia, they indicate that if procaine is indeed "fixed" in the nerve tissue the total amount necessary must be relatively small. F. Howarth and E. R. A. Cooper[6] go further and state simply that there is neither convincing proof nor accepted definition of "fixation".

The problem still remains of the route by which substances pass from the theca to the systemic circulation, a problem bound up with theories regarding the circulation of cerebro-spinal fluid. The most widely accepted theory, that put forward in Best and Taylor's book, "The Physiological Basis of Medical Practice", is that the main absorption of fluid is through the arachnoid villi into the great venous sinuses into which these villi project. The mechanism is considered to be one of filtration. "True solutions readily pass through the arachnoid villi, colloids more slowly, the rate depending upon the size of the molecule; particulate matter does not pass." Passing reference is made by Best and Taylor to the occurrence of some absorption in the spinal canal, but this is apparently not considered to be of great importance; mention is made also of experimental reversal of flow of cerebro-spinal fluid during the intravenous injection of hypertonic saline and the obstruction of the aqueduct of Sylvius, dye having been seen to pass from the ventricles into the capillaries of the chorioid plexus. The two main routes of absorption other than via the arachnoid villi which have had support are via the lymphatic drainage

[1] *The Journal of the American Medical Association*, December 3, 1932.
[2] *The Journal of the American Medical Association*, July 30, 1949.
[3] *The Lancet*, October 8, 1949.
[4] *Archives of Surgery*, January, 1943.
[5] *The American Journal of Surgery*, July, 1929.
[6] *The Lancet*, November 19, 1949.

of the spinal subarachnoid space and via the local venous drainage of the spinal theca. The lymphatics have apparently had few champions, but there are those who feel that direct local venous drainage has not been given the importance it warrants. Weight to this viewpoint has been added by Howarth and Cooper, to whose paper reference has already been made. First of all they introduced radioactive dibromoprocaine (and subsequently other radioactive substances) into the lower part of the spinal arachnoid space of cats, and withdrew samples of cerebro-spinal fluid from the lumbar region and from the *cisterna magna* at succeeding intervals. The concentration of the substances dropped rapidly in the lumbar cerebro-spinal fluid, but in no case was a concentration found in the cisternal fluid to account for this decrement in terms of an upward circulation. Next they obtained samples from veins most likely to drain the subarachnoid space. Dibromoprocaine appeared in the venous system almost immediately on its injection into the spinal theca, the azygos vein carrying a much greater concentration than any other vein; radiosodium, radiophosphorus and radiobromine appeared to behave similarly. The time interval was strongly against any possibility that the substances had reached the circulation via the arachnoid villi. Precipitate generalizations from these animal experiments cannot be made, but they are highly suggestive, and the occurrence of rapid removal of substances by direct venous drainage from the spinal theca would explain why some investigators have found relatively low concentrations of penicillin in the *cisterna magna* after relatively large concentrations of the drug have been introduced into the lumbar subarachnoid space; it would also partially explain why the medullary centres are not paralysed in patients subjected to spinal anæsthesia.

Finally, although it further confuses rather than clarifies this difficult subject, mention must be made of contentions brought forward last year by G. B. Hassin.[1] Hassin is completely at variance with the current concept of the circulation of the cerebro-spinal fluid. He considers that the fluid does not originate in the chorioid plexuses, but that it represents the tissue fluids of the brain and is formed by transudation from the brain into the tissue spaces of Virchow-Robin; it is discharged into the cerebral ventricles and subarachnoid spaces and functions as lymph to carry away the waste of the central nervous system. The function of the chorioid plexus, according to Hassin, is to excrete, to eliminate from the fluid those substances which might be injurious to the organism or interfere with its absorption from the perineurial or root spaces (which is what Hassin believes takes place); such substances are picked up as lipoids, lecithin, pigments and foreign substances (as fragments of *Torula histolytica*, carbon *et cetera*). With regard to circulation of the cerebro-spinal fluid, Hassin states that there is no driving mechanism for active circulation and that such circulation as is described occurs after manipulations such as lumbar punctures. So he adds yet another voice to what is already something of a Babel; but perhaps even Babel is to be preferred to a single voice speaking without authority.

[1] *Journal of Neuropathology and Experimental Neurology*, April, 1948; quoted in "The 1948 Year Book of Neurology, Psychiatry and Neurosurgery".

PROGNOSIS OF ACUTE MYOCARDIAL INFARCTION.

THE voluminous literature about coronary arterial disease and its associated accidents has of recent years been concerned a good deal with the refinements of diagnosis. The electrocardiograph has proved even more valuable than was thought at first in determining the location of an infarction and in detecting evidence of coronary insufficiency, and in this way has contributed to knowledge concerning prognosis. But any single attack is always an anxiety, for it is difficult to assess the importance of the signs and symptoms in terms of the future. Therefore it is useful to consider the purely clinical information gathered from a series of acute coronary occlusions to see what help such a study gives in making a forecast of the patient's prospects. A series of 240 cases of acute myocardial infarction has been analysed by F. T. Billings, B. M. Kalstone, James L. Spencer, C. O. T. Ball and G. R. Meneely; they have used the data readily available to any physician carrying out routine observations at the bedside.[1] The patients included in this study were selected from a larger series of 591 patients admitted to the Vanderbilt University Hospital from 1925 to 1946. The 240 case histories chosen satisfied certain criteria, including the familiar clinical picture of pain not due to overdemand on the circulation and not relieved by nitrites or rest, signs of shock, rise in heart rate and fall of blood pressure, fever, leucocytosis and acceleration of the sedimentation rate. In addition characteristic electrocardiographic changes or demonstration of infarction at autopsy were available as evidence. Without one or more of these criteria the records were rejected from the series. Only one attack was analysed in any given case. The majority of patients studied were seen in their first attack. Only data of statistical significance were used in the study. "Immediate" mortality was arbitrarily taken as meaning the death rate due to the infarction within thirty days of its occurrence; this was found to be 40·4%. In first attacks the percentage of deaths was 36·4, while in second and third attacks it was 53·6. The effect of age is difficult to assess; there were more deaths relatively when the age passed fifty years. No racial or seasonal influence was detected. The debatable point of physical activity at the time of an attack was not settled, for although in 64% of patients there had been no activity at the time of the attack, the authors give no information concerning the degree of activity for various periods before the onset. Twenty-two *per centum* of patients had been vigorously active at the time when acute symptoms began. It is noted by the authors that not every patient who has coronary disease leading to infarction experiences symptoms. The question of pain is also interesting. No mention is made of those symptoms which may be termed "substitute" symptoms for pain, such as dyspnœa. It has been pointed out by a number of clinical observers that it is unusual for more than one symptom of importance or severity to occupy the field of consciousness at the same time. Nevertheless the presence or absence of pain is most important, as many have shown; the authors found that absence of pain is of sinister import. It is understood that the word "pain" includes such forms of discomfort as thoracic oppression, even though the patient did not actually employ the usual descriptive word. The past history was also of importance. Anginal attacks were recorded in about half the histories. Hypertension was equally common, but had no intrinsic effect on the death rate, though this has not been the experience of all writers. Congestive failure, on the other hand, was of great importance in the past history and introduced a more serious outlook on the future. The degree of fever found during the acute attack was of value in prognosis; with the exception of those patients who died quickly with subnormal temperature and other signs of shock, the higher the temperature, the greater the risk of early death. A pulse rate of below 60 per minute or over 110 was found to be ominous. As might be expected, signs of cardiac dysfunction, such as gallop rhythm, very weak sounds and arrhythmias, are usually correctly looked on as important; though Billings and his associates did not find any special significance in gallop rhythm, they found it frequently when congestive failure was present, so that it cannot be disregarded.

To summarize, this question may be asked: Is it of importance in trying to forecast a patient's prospect after an acute infarction of the heart to observe the temperature, the pulse rate, the nature and regularity of the heart sounds and the presence of leucocytosis, and to ascertain if any evidence of cardiac dysfunction existed before the attack? The answer is certainly "yes", for these simple observations, though in the main obvious, contribute to that combination of careful examination, memory of past experience, and common sense which is called clinical judgement.

[1] *The American Journal of Medicine*, September, 1949.

Abstracts from Medical Literature.

SURGERY.

Thoracic Complications of Amœbiasis.

ROBERT SHAW (*Surgery, Gynecology and Obstetrics*, June, 1949) states that of the general population of America 20% are infested with *Entamœba histolytica*. Hepatic abscess will complicate 5% of such infestations and the thoracic cage will be involved in 15% of patients with liver abscesses. Amœbic abscess occurs fifteen times more commonly in men than in women; the majority of those affected are aged between twenty and fifty years. The hepatic abscess is usually single and in the right lobe of the liver. Thoracic complications arise from spread into the pleural cavity or into the lung. Abscesses of the left lobe may extend into the pericardial sac. Solitary amœbic abscesses have been described; they are rare and probably embolic. In half the cases with pleuro-pulmonary complications, there is no history of dysentery. The usual complaint is of pain in the right side of the chest and shoulder, of an unproductive cough and later of hæmoptysis, which precedes the anchovy paste sputum. There is a septic fever and the usual evidences of infection appear. A radiograph characteristically shows the localized elevation of the diaphragm, which is best seen in the lateral view. If there is extension into lung, a shadow is seen involving the lower lobe and tapering to the hilum, with or without the signs of an abscess; if into the pleural space, there will be the signs of pleural exudate. Loculation is common with amœbic empyema. The mortality is high, less when the lung is involved without an empyema and least when a broncho-hepatic fistula has occurred without empyema or pulmonary abscess. Emetine is given, one grain intramuscularly for six to ten days. Aspiration of the liver abscess should be done if possible. Surgery is indicated in pleuro-pulmonary complications when secondary infection has occurred, when an empyema is present and does not respond to aspirations, when a broncho-hepatic fistula persists, or when pulmonary fibrosis consequent on the abscess is producing symptoms.

Acute Mesenteric Lymphadenitis.

R. W. POSTLETHWAIT AND F. H. CAMPBELL (*Archives of Surgery*, July, 1949) have investigated the records of 1604 patients admitted into Duke Hospital with symptoms and signs of an acute condition in the abdomen which led to treatment by appendicectomy. Of these 931 had acute appendicitis, and of the remaining 673 patients, 268 with a normal appendix had enlargement of mesenteric lymph nodes. In these cases, the diagnosis was made of non-specific mesenteric lymphadenitis, a symptom-complex characterized by abdominal pain and tenderness. For the patients suspected of having this condition, the presence of acute appendicitis cannot be excluded by the history, by the physical examination or by the laboratory findings. Because of this difficulty in differentiation, with rare exceptions, operation is urgently indicated when either diagnosis is made. Little is known of any ætiology, and there is some difference of opinion as to whether the diagnosis can be made pre-operatively and with wisdom laparotomy not be performed. The sex incidence in the authors' series was just less than two to one in favour of females, and the age incidence showed a comparative increase in the lower age groups. Abdominal pain and right lower quadrant tenderness were present in mesenteric lymphadenitis nearly as commonly as in acute appendicitis, and the temperature on the patient's admission to hospital was generally higher for those with appendicitis. The authors consider that the enlarged lymph nodes represent reaction to injurious agents, bacteria or viruses or their toxins which originate in the appendix or ileum. Mesenteric lymphadenitis is an operative diagnosis, and can be strongly suspected pre-operatively, but a pre-operative diagnosis of mesenteric lymphadenitis should necessitate operation as readily as a pre-operative diagnosis of acute appendicitis.

Surgical Treatment of Patent Ductus Arteriosus.

W. J. POTTS (*Surgery, Gynecology and Obstetrics*, May, 1949) states that while it is only ten years since Gross successfully ligated a patent *ductus arteriosus*, operation for this condition has now become an accepted surgical procedure. However, there is still a considerable difference of opinion as to the best and safest method of closing the patent ductus. The author discusses the attitude of surgeons, who favour either the ligation or the division of the ductus. He himself has operated on 22 patients, successfully using ligation; however, he believes that the preferable operation is division and suture. He describes in some detail the postero-lateral incision and approach, and the method of division and suture. He uses special ductus clamps, which, he states, successfully hold the divided ductus stumps without damaging them.

Splenectomy.

W. H. COLE, L. WALTER AND L. R. LIMARZI (*Annals of Surgery*, May, 1949) discuss the indications for and results of splenectomy in the light of 87 cases in which splenectomy was performed with an operative mortality rate of 8%. They state that although much progress has been made in the diagnosis and treatment of splenic disease, there is still much controversy, particularly on the indication for splenectomy. The functions of the spleen are but incompletely known, and more is known of the adverse physiological effects of the diseased spleen on the organism than the functions of the normal spleen. The authors discuss what is known of the normal and the abnormal functions of the spleen and then the effects of its removal. They differentiate between congenital familial (microcytic) hæmolytic anæmia and acquired secondary (macrocytic) hæmolytic anæmia; results of splenectomy in the familial type are consistently good, but are unsatisfactory in the acquired type. Again in thrombocytopenic purpura, it is essential to distinguish between primary (Werlhof's) and secondary or symptomatic purpura, since splenectomy is of little or no value in the latter type, which may be secondary to drugs, leuchæmia, aplastic anæmia, radiation, tumours, infection *et cetera*. Bone-marrow study is diagnostic, revealing a marked increase of immature megakaryocytes without platelet formation, due presumably to an inhibiting effect of the spleen on their maturity and release from the bone marrow into the blood-stream. Indeed, the number of megakaryocytes in the bone marrow is such a reliable indication of true thrombocytopenic purpura, that it may be used as an index of prognosis following splenectomy. Opinions vary considerably as to whether or not splenectomy may have curative or only remedial effects on the disease in early cases. The authors cannot determine whether thrombosis or anomalous obstruction of the splenic vein should or should not be included in Banti's disease, but they are convinced that splenectomy in the early stages of splenic-vein obstruction should be curative. Splenectomy is indicated in the syndrome described as Felty's disease. Although controversy still exists as to whether this syndrome is a separate entity, the authors classified five cases as such, as they could not be classified into any other group. Splenectomy is also indicated in the two rare conditions of splenic neutropenia, and primary splenic panhæmatocytopenia. Rupture of the spleen is, of course, an indication for removal of the spleen, and cysts, primary tumours and abscesses are generally an indication. Splenectomy is indicated in Gaucher's disease generally only when panhæmatopenia develops. Malignant tumours of the cardiac end of the stomach, ptosis of the spleen and malaria are mentioned as occasional indications.

Lactation after Thoracoplasty and Pneumonectomy.

D. SALKIN AND E. W. DAVIS (*The Journal of Thoracic Surgery*, August, 1949) report ten cases of lactation in non-puerperal women following thoracic operations; in nine of the cases the operation performed was extrapleural thoracoplasty and in the tenth pneumonectomy. The authors believe that the evocation of the lactogenic hormone which usually results from the mechanical stimulation of the nerve fibres of the breast by the act of suckling was supplied in these cases by the operation. They think it possible that the lactogenic hormone may normally circulate at some time during the menstrual cycle.

Congenital Atresia of the Œsophagus.

I. A. BIGGER (*Annals of Surgery*, May, 1949) discusses the classification, diagnosis and treatment of congenital atresia of the œsophagus and presents the results for eighteen patients on whom he has operated. He states that the diagnosis of the condition can be made with a considerable degree of certainty on the basis of excessive drooling of saliva and immediate regurgitation of everything given by mouth, with strangling. A soft rubber catheter passed into the œsophagus will meet an obstruction from 10 to 12 centimetres from the gum margin. If there is evidence of gas in the stomach and intestines, there is almost certainly an associated tracheo-œsophageal fistula. A definite diagnosis can be made by X-ray studies following the injection of iodized oil into the upper part of the œsophagus. Barium should never be used for this purpose, for if it is

regurgitated and aspirated into the tracheo-bronchial tree, it produces obstruction of many of the smaller bronchi and leads to serious respiratory difficulties. It is of great importance to determine the presence or otherwise of a fistula and the distance between the two segments when there is a fistula. The author does not favour tracheoscopy recommended by Byron because of the danger of producing œdema of the trachea. He uses the injection of a little iodized oil into the trachea as well as the œsophagus when in doubt as to a small fistula. It is only within the last ten years that the treatment of œsophageal atresia has been altogether successful. The author uses a right extrapleural approach after preparation by rehydration and the use of blood. The operation is performed under cover of penicillin before and after operation and streptomycin after operation. The details of the operative technique are given and the post-operative care is discussed. From 1941 to 1948, the author operated on eleven infants with congenital atresia of the œsophagus with tracheo-œsophageal fistula. Only two of the eleven survived and one of these died at the age of seven months after an operation for the repair of a recurrent tracheo-œsophageal fistula. In 1948 seven infants were operated on and all have recovered. In all seven cases, gastrostomy was performed one or two days before thoracotomy and the gastrostomy tube was used to deflate the stomach and intestines. Because of this, there was decided improvement in the ease of administration of the anæsthetic, and in every instance the infant left the operating theatre in good condition. In five cases the œsophageal segments were of sufficient length to permit satisfactory anastomosis. In the other two, because of a short proximal œsophageal segment, the fistula was closed and cervical œsophagostomy was carried out. In one case an end-to-side anastomosis has been made between the cervical part of the œsophagus and the fundus of the stomach, through a subcutaneous tunnel. The second infant will not undergo œsophago-gastrostomy for several months. The author is of the opinion that the improvement in the results obtained in the later group is due to the use of the preliminary gastrostomy, as well as the use of penicillin and streptomycin.

The Small Stoma in Partial Gastrectomy.

H. W. PORTER AND Z. B. CLAMAN (*Annals of Surgery*, April, 1949) have used a small stoma after partial gastrectomy for ulcer in an endeavour to improve the results. From a study of the literature they concluded that although the size of the stoma did not modify the quantitative emptying time, the presence of sphincteric action at the jejunal stoma did delay emptying. Gastroscopic examination has revealed that where sphincteric action is present there is less jejunitis. The authors made a stoma 2·5 centimetres in length in a series of 118 gastrectomies and claim the following advantages: (i) the advantage of the Hopmeister-Finsterer valve—a decreased incidence of leakage at the upper end of the anastomosis, and a decreased incidence of retrograde influx into the proximal jejunal loop; (ii) prevention of bulging of the jejunum after food, a condition which prevents the patient from eating a normal volume of food and is a cause of post-prandial discomfort; (iii) reduced risk of post-operative hæmorrhage, as the length of the exposed incision is less; (iv) increased emptying time, the average time for the fluid level in the gastric remnant to disappear (quantitative emptying time) being fifty-eight minutes; (v) the sphincter-like action of the stoma and the decrease in jejunitis. With these advantages the authors claim that a normal diet may be commenced much earlier with considerable benefit to the psychological and physical well-being of the patient. The dumping syndrome has been present in 15% of cases as opposed to 35% in the usual series. No evidence of stenosis of the stoma has been found over five years.

The Synovial Membrane of the Knee in Pathological Conditions.

R. SOBUR (*The Journal of Bone and Joint Surgery*, April, 1949) has studied the reaction of the synovial membrane of the knee in various pathological conditions. He describes in detail the findings from examination of material obtained at operations on the knee joint and also the results of animal experiments. He considers that this study permits him to define the cellular behaviour of the synovial membrane to various irritants. In meniscal tears it is of a mechanical nature, in hæmarthroses it is of a chemical nature (endogenous proteins), and in villous arthritis it is of an allergic nature (phenomenon of Arthus). The histological picture of each appears so different and so characteristic that it has a certain value in pathogenic research on chronic affections of the knee. It will be possible in the future to establish with precision, through a biopsy of the infrapatellar fat pad, the factor responsible for the origin of a morbid process.

Cancer of the Naso-Pharynx.

M. W. SIMMONS AND I. M. ARIEL (*Surgery, Gynecology and Obstetrics*, June, 1949) report on 150 cases of cancer of the naso-pharynx. The patients represented 0·7% of all patients admitted to a veterans' hospital with malignant tumours. They formed 11·5% of all with intraoral cancer. No significant predisposing factors were found. The lesion usually manifested itself as a slight bulge or ulceration about the fossa of Rosenmueller. No lesion might be manifest for one month to two years after the appearance of massive cervical metastases. In 40% of cases the exact location of the origin could not be determined. The growth was composed of squamous cells in one-third of the cases and transitional cells in one-third. One growth in ten was anaplastic. Extension to the base of the skull with affection of the cranial nerves by pressure was present in 37% of cases, the abducent, the hypoglossal and the trigeminal being the nerves most commonly involved. The auditory nerve was involved in only 2·6% of cases. In 87% of cases cervical metastases were present, half of them being bilateral, usually into the upper deep cervical lymph nodes. Half of the patients coming to autopsy had generalized metastases. The patients usually presented with symptoms due to the extension of the growth, for example, swelling in the neck, pain in the side of the face and head. Only 4% presented with nasal obstruction. The diagnosis on admission to hospital was more often wrong than right, the chief errors being inflammatory adenitis, Hodgkin's disease, primary cervical carcinoma and cerebello-pontine angle tumour. Usually seven months elapsed between the first consultation and the final diagnosis. Treatment was by irradiation, given daily through two ports till a total of 7000*r* had been reached. Separate portals were used for the cervical metastases. The five-year survival rate was 9·8%.

Intractable Pain Treated by Arteriectomy.

N. E. FREEMAN *et alii* (*American Heart Journal*, September, 1949) have successfully revived an old method of treatment for the severe rest pain with paræsthesia and numbness of the extremity which may occur after recovery from acute occlusion of a major artery to the extremity, even though the circulation is sufficient for tissue nutrition. The condition is sometimes termed "ischæmic neuritis". Temporary relief may be obtained by blocking the sympathetic nerves to the leg, but sympathectomy is contra-indicated because it may precipitate gangrene. The authors, following R. Leriche, have located the site of the occlusion by arteriography and excised that part of the artery in ten patients. Immediate relief of pain followed, though there was no significant increase in the circulation. It was supposed that the operation interrupted some nervous reflex which originated in the thrombosed artery.

Polyostotic Fibrous Dysplasia: Albright's Syndrome.

C. K. WARRICK (*The Journal of Bone and Joint Surgery*, May, 1949) reports four cases of polyostotic fibrous dysplasia; this condition may, in addition to the features which characterize Albright's syndrome, namely, the association of predominantly unilateral skeletal changes with pigmentation of the skin and with precocious puberty in females, be associated with other endocrine changes. In two of the four cases—all the patients were males—secondary sex characters developed during the first decade. The affected bones have a "ground glass" appearance, in which homogeneous areas of increased or decreased density are present with thinning of the overlying cortex. The base of the skull often shows enormous hypertrophy of dense bone, which may obliterate the nasal sinuses and compress the cranial nerves. No constant changes are found in the chemical constitution of the blood other than elevation of the alkaline phosphatase level if the bone changes are widespread. The dysplasia is often confused with parathyreoid osteodystrophy, and sometimes the parathyreoid glands are needlessly explored. This confusion should not arise if it is remembered that no general skeletal decalcification, and no constant changes in the blood calcium or phosphorus content, occur in polyostotic fibrous dysplasia. The radiographic appearances of healing parathyreoid osteodystrophy are, however, indistinguishable. In addition the dysplasia may be confused with Paget's disease, Ollier's disease, *osteogenesis imperfecta tarda*, and the lipoid reticuloses. No effective therapy has been discovered for this disease. Pathological fractures and deformities may require treatment.

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held at the Children's Hospital, Carlton, Victoria, on August 10, 1949. The meeting took the form of a symposium on pulmonary tuberculosis in childhood. DR. K. G. KERR read a paper entitled "A Tuberculosis Survey of Children in a Provincial City" (see page 901).

Clinical Manifestations of Primary Pulmonary Tuberculous Infection in Childhood.

DR. L. P. WAIT discussed modes of presentation, investigation, prognosis and treatment in relation to infants and children with primary pulmonary tuberculous infection.

Considering symptomatology first, Dr. Wait stated that there was no one common clinical pattern of tuberculous infection in childhood. The child with primary pulmonary tuberculous infection might be either very well or very ill and there might be all grades of ill health between those two extremes. Many children with tuberculous infection presented no symptoms of general ill health and no pulmonary symptoms. The commonest clinical evidence of infection was vague general ill health with malaise and anorexia, and any child who was so afflicted should be suspected of having tuberculosis. An infective illness of a subacute type might also be an early manifestation of the infection. At times the symptoms were predominantly pulmonary in nature and symptoms of general ill health might be absent or minimal. More commonly there was a history of a respiratory tract infection which failed to clear up satisfactorily, and such an illness might be diagnosed as bronchitis. An irritating or chronic cough might be the initial symptom and occasionally asthmatical breathing might herald compression of some portion of the tracheobronchial tree by enlarged mediastinal or hilar lymph glands. Phenomena depending on the so-called allergic manifestations might declare the onset of the tuberculous infection; *erythema nodosum*, phlyctenular conjunctivitis, polyarthritis resembling rheumatic fever, or a pleural effusion might cause difficulty in establishing the diagnosis. Hæmatogenous disseminated lesions in other tissues might provide the first factor that drew attention to a pulmonary infection, for example, tuberculous meningitis, or bone and joint tuberculosis. So protean were the clinical manifestations that the clinician must be ever conscious of the possibility of tuberculous infection in patients presenting any of the features mentioned.

In the majority of patients there were no abnormal physical signs on clinical examination for the reason that the primary focus in the lung was usually very small and enlargement of the hilar glands was not detectable by clinical physical methods. The Mantoux test, with 1:1000 dilution of old tuberculin, was the most reliable indicator of infection, and that test was always the first method to be used on any child suspected of the infection. Radiological examination with a careful technique should then be carried out, and finally culture of the fasting gastric contents of those patients who gave a positive result to the Mantoux test and had either constitutional or radiological evidence of infection.

With regard to prognosis, the early months following the primary infection were the most dangerous for the patient, for it was in those early months that hæmatogenous dissemination, which not infrequently resulted in either tuberculous meningitis or a miliary infection, was likely to occur. The younger the child the greater that danger, and it was well to remember that the majority of fatal infections occurred within the first two to three years of life. Acute infections, especially measles and whooping-cough, were a potential danger to the child with a primary infection as a flare-up of the primary lesion might occur. It had to be stressed, however, that the prognosis for the majority of patients was good provided reasonable treatment was available, and the child was not subjected to further infection.

Discussing treatment, Dr. Wait said that as there was a strong tendency for the primary lesion to heal, an adequate period of rest materially assisted that process. That rest period always was the basis of treatment. Sanatorium treatment was not necessary for the majority of the children and was undesirable, provided reasonable home conditions were obtainable. Small children, especially, should never be placed in an institution for a period of many months until all attempts to provide care in a home had been exhausted. Contact with the infecting source must be controlled; usually that could be attained only by removal of the infected adult to a sanatorium. Measles and whooping-cough were potentially dangerous diseases for those children and protection was advisable, by the use of γ globulin or pooled human serum in the former and immunization in the latter. Streptomycin was rarely indicated for a primary infection, and it should be reserved for such disorders as miliary tuberculosis and local spread of the primary tuberculous complex.

Dr. Wait illustrated his talk with clinical examples of patients who had been under his care at the Children's Hospital.

Pathology of Pulmonary Tuberculosis in Childhood.

DR. REGINALD WEBSTER stated that a discussion of pulmonary tuberculosis in childhood resolved itself into a consideration of primary tuberculous infection as it manifested itself in the lungs of children. It must, of course, be allowed that occasionally in older children the tuberculous process in the lung had the morbid anatomical features of the lesion of reinoculation, or the so-called adult type of pulmonary tuberculosis, but he proposed to restrict his observations to primary infection. He considered that his function was to sketch in, with as few strokes as possible, the background of morbid anatomy and pathogenesis against which all other aspects of the subject should be considered.

Primary infection, in which the tubercle bacillus established itself in the virgin soil of the tissues of an individual, child or adult, who had never before made contact with tuberculosis, might be acquired via the lung, the tonsil, the alimentary tract or the skin. It was Dr. Webster's experience that the cutaneous route of infection merited more notice than was generally accorded it in text-books, and to dismiss inoculation tuberculosis as confined in its incidence to those unfortunate enough to follow the hazardous occupations of cleaners of sputum cups, butchers and pathologists, was to do that aspect of tuberculous infection less than justice. Wherever the primary lesion was located it was followed by a rapid and conspicuous enlargement of the immediately related lymphatic glands. The primary focus, with the enlarged and caseating glands which had derived their infection from it, together constituted the primary complex.

A notable feature of the primary complex was that the initial lesion, the lesion of entry of the tubercle bacillus, was often overshadowed by the glandular component. That characteristic of primary infection was well illustrated by children who presented with tuberculous cervical adenitis. Tuberculous glands of the neck were referable to a primary lesion in the tonsil, and although Dr. Webster had on very many occasions demonstrated tuberculosis of the tonsil, by histological section or cultivation of *Mycobacterium tuberculosis*, on the same side as the enlarged and grossly caseating glands of the neck, he could not remember an instance in which the tuberculous affection of the tonsil was apparent on macroscopic inspection.

Dr. Webster said that the same assumption of predominance on the part of the lymph gland participating in the primary complex was formerly a common observation in infection via the intestine, when tuberculosis due to the bovine type of *Mycobacterium tuberculosis* was of more frequent occurrence than at present. It had often been his experience in the past to find enlarged and caseating mesenteric glands, and to be obliged to search for the small lesion of entry in the lymphoid tissue of the intestine—sometimes indeed without success, the small primary focus having healed. The position was similar with inoculation tuberculosis, or infection via the skin. He well remembered two children, who presented with inguinal swellings, from which pus containing numerous tubercle bacilli was evacuated. One child had a small sore, mistaken for impetigo, on the medial aspect of the thigh, and the other an apparently insignificant lesion on the medial aspect of the great toe. Both were excised and proved tuberculous, and they were actually overlooked until the demonstration of tubercle bacilli in the inguinal cold abscess indicated a search for the primary focus.

The extent to which the tracheo-bronchial lymph nodes were involved in primary tuberculous infection of the lung was a feature of the morbid anatomy of far-reaching implications. Not only were the nodes so affected a menace from the point of view of metastatic infection, but, even though the tuberculous process might recede to the degree represented by the conversion of the nodes into a fibrotic matrix for the accommodation of calcareo-caseous spicules, such apparently burned-out lesions could not with safety be assumed to be devoid of viable tubercle bacilli. It was generally agreed that the tracheo-bronchial lymph nodes harboured tubercle bacilli more often than any other part of the body, and if endogenous reinfection should occur in

the later life of a child in whom primary infection had followed its usual benign course, the bacilli of reinoculation were much more likely to emanate from the glandular than from the pulmonary constituent of the primary complex. Primary infection was of necessity exogenous; reinoculation might be exogenous or endogenous, and Dr. Webster did not propose to broach the controversial subject of the source of the bacilli of reinoculation further than to say that the relative frequency with which they came from without or within was one of the many problems in tuberculosis in which finality had not been reached.

In the beginning the primary focus was a small granuloma—it might appropriately be called a chancre—about the size of a bean or smaller, with caseous centre, sometimes encircled with minute tubercles and on other occasions by fibrous tissue, according to the age of the lesion and the progress it had made towards recession and healing. A very important, indeed essential, characteristic of the primary focus in the lung was that it displayed no predilection for any particular lobe of the lung. It might be located anywhere in the superficial pulmonary parenchyma, from apex to base, on medial, lateral or diaphragmatic surface. In that respect it contrasted sharply with the lesion of reinoculation, which exhibited with remarkable constancy a predisposition for the apical portion of the lung, or, more accurately, the upper third of the upper lobe.

Another well-sustained feature of the primary focus in the lung was its peripheral location; it would almost always be found abutting on the pleural surface, or, at all events, not far beneath it. Generally single, the primary lesion might occasionally have more than one focus within a small radius, and not uncommonly a streak of minute subpleural tubercles indicated the path by which infection had spread to the tracheo-bronchial glands.

Dr. Webster said that it might be appropriate at that stage to summarize the morbid anatomical features of primary tuberculous infection of the lung, displayed most characteristically in the child, and contrast them with the lesions of reinoculation, which were generally deferred until adult life. In the child progressive lesions were initiated anywhere in the lungs; in such lesions caseation predominated and fibrosis was not a feature; the regional lymph nodes were involved to a degree which frequently rendered the pulmonary lesion relatively insignificant. In the adult the disease process arose almost invariably in the upper portion of the upper lobe of the lung, and whilst there was no lack of caseation, concomitant fibrosis was as a rule conspicuous; the regional lymph nodes, even in the presence of destructive pulmonary lesions, often appeared normal on macroscopic inspection. Metastatic lesions, of course, such as miliary tuberculosis and tuberculous meningitis, occurred much more commonly in the child than in the adult.

The course taken by the tuberculous infection after the establishment of the primary complex depended on a number of factors, chief among which were the age of the child, his environmental and nutritional conditions, and what Dr. Webster termed his genetic credit balance of native resistance to tuberculous infection. Age and environment were considerations of first importance. The younger the child (or infant) and the longer he was left in the tuberculous household in which, in the majority of instances, he had acquired his tuberculosis, the more likely was the lesion to progress, with extension of the small initial lesion to a caseating pneumonia of varying extent, or perhaps bronchogenic spread leading to tuberculous bronchopneumonia, which might be widely distributed throughout the lung, or, a very common climax, terminal hæmatogenous sowing and miliary tuberculosis.

If a broad view was taken of the problem of tuberculosis in childhood the prospect was much brighter. For a child who contracted infection after the age of five years, particularly if he could be segregated from the phthisical relative in the home from whom he had in all probability acquired his infection, the outlook was good. Given reasonable opportunity, the natural tendency in older children was for the Ghon lesion to heal, become calcified, or even become ossified, at which stage it might be very difficult to find by dissection of the lung, but would reveal itself as a small radioopaque spot on an X-ray film. In a proportion of older children, however, the primary lesion was progressive: encapsulation of the initial lesion failed, or caseation spread into a bronchus from tuberculous glands in the hilum, and there followed intrabronchial spread; erosion of a blood vessel included in or contiguous with a caseating lesion undergoing softening, or the discharge of its softened contents by a caseating intimal lesion in a vessel such as the pulmonary vein, was fraught with serious risk of overwhelming hæmatogenous dissemination. That dire train of events was more likely to ensue on primary pulmonary tuberculous infection established before the age of three years, but at any time in childhood it was likely to be initiated by acute respiratory infection, such as that involved in an attack of measles, whooping-cough or severe influenza. In many a child harbouring a static primary complex, miliary tuberculosis had followed an attack of measles or whooping-cough, the *modus operandi* being that intense vascular engorgement and capillary damage, occasioned by acute inflammation of the tissues in the immediate neighbourhood of a tuberculous focus, permitted escape of bacilli into the blood-stream; or it might be that dissemination was by lymphatic channels as had been so well expressed by the late A. K. Krause: "Exalt the circulation (and therefore the flow and content of lymph) in the affected region, and you promote the sending over to the body of intrafocal poisons, as well as the outsweep of bacilli which had been more securely interned before."

A phenomenon of particular interest associated with primary infection was that which had been designated by the term epituberculosis. It consisted essentially in a benign lesion which occurred in children who reacted to tuberculin, and was represented in a radiogram by a large, dense, homogenous shadow of characteristically sharp outline; the shadow commonly obscured a wedge-shaped area of pulmonary parenchyma, which might extend from the hilum of the lung to the pleura. Such an opacity was seen to resolve gradually under radiological observation, though the clearing process might take several months, or even a year, possibly longer. The relative lack of symptoms in the presence of such alarming shadows, and the gradual restoration of radiolucency, were the distinctive and arresting features of epituberculosis.

What was the pathological basis for the clinical and radiological entity known as epituberculosis? Although Dr. Webster had never pretended to more than the small Latin and less Greek attributed to Shakespeare by Ben Jonson, when he first became acquainted with the term epituberculosis he remembered that the Greek prefix "epi" was applied as the equivalent of upon, at, on the ground of, or in addition. Hence the term epituberculosis conveyed to him an impression of a process associated with, or grafted upon, a tuberculous lesion, but not necessarily itself tuberculous; or in other words, a non-specific pneumonic exudate or consolidation prone to occur in the vicinity of a tuberculous focus. Some such meaning was no doubt intended by the sponsors of the term epituberculosis, but evidence had accumulated that in very many instances epituberculosis so-called was a specifically tuberculous process, and in short represented nothing less than a resolving tuberculous pneumonia.

It seemed incredible to many, of whom for many years he had been one, that so extensive a pneumonia, if tuberculous, could resolve in the manner characteristic of the exudate of epituberculosis. But case records such as that of Rubenstein, as cited by Arnold Rich, and that published in the *Archives of Disease in Childhood* by Professor J. C. Spence, in that they demonstrated the tuberculous nature of the exudate by biopsy investigation, did much to break down the conservatism of those who found it difficult to accept the view that the basis of the radiological and clinical entity which had become known as epituberculosis was a resolving tuberculous pneumonia.

The child concerning whom Rubenstein reported his observations exhibited a shadow in the thoracic radiogram typical of epituberculosis. Empyema was suspected and an operation performed. Instead of an empyema there was found a large wedge of pulmonary tissue consolidated to a degree which gave rise to suspicion of a malignant neoplasm. A biopsy specimen was secured, which in microscopic sections showed lung tissue consolidated by aggregated epithelioid cells, giant cells, lymphocytes and fibroblasts. Subsequently to the operation the consolidation was observed to clear gradually, in serial radiographs taken over a period of a year.

In that report it was not recorded that tubercle bacilli were shown to be present, but the demonstration of *Mycobacterium tuberculosis* was the feature which placed Spence's report beyond criticism. There again there was an extensive, wedge-shaped shadow in the radiogram; it was situated in the upper lobe of the left lung of an infant, aged seven months, who was found to react to tuberculin. Two lung punctures were performed, four weeks apart, at different sites in the lesion, and both yielded caseous material in which tubercle bacilli were demonstrated. Three years later the child was quite well and no longer displayed the initially startling radiographic shadow.

Professor Arnold Rich, in his standard work on the pathogenesis of tuberculosis, had recorded autopsy details, with illustrative anatomical preparation, which demonstrated convincingly that extensive pulmonary lesions, induced by the aspiration of tuberculous material from caseous lymph nodes that discharged into bronchi, could heal completely.

As a rule, when a caseous lymph node discharged into a bronchus, the spread of infection led to progressive and fatal disease, but it was possible that the consolidation supervening on that generally disastrous event might under certain conditions undergo resolution and healing. If the aspirated material was rich in viable bacilli, the chance of overcoming the infection must be slight. If living bacilli were not too numerous, the resistance stimulated by the primary infection might conceivably effect their destruction. It was the experience of every pathologist that caseous material was often very poor in tubercle bacilli; the discharge of a caseous lymph node into a bronchus therefore did not necessarily mean the inoculation of the lung with large numbers of aspirated bacilli. Even were there no living bacilli, the aspiration of caseous material as such, with its content of dead bacilli and their fragments, and tuberculo-protein from disintegrated bacilli, could well result in pulmonary consolidation in an individual rendered hypersensitive by the primary infection. It had been shown that bacillus-free extracts of tuberculous lesions possessed strong tuberculin activity, and also that the introduction of tuberculo-protein into the lung of the hypersensitive body by way of the bronchi induced a pronounced pneumonic consolidation, which might heal completely.

Time did not permit more than a passing reference to the work of Oppenheimer, who, working in Rich's laboratory, succeeded in inducing a faithful reproduction of epituberculosis, as viewed both radiologically and clinically, by the introduction of heat-killed tubercle bacilli into the lungs of hypersensitive animals by way of the bronchus. When the killed bacilli were planted in the bronchi of normal non-hypersensitive animals, the resulting reactions in the lungs were too slight to be recognized at any time in the radiographs.

Dr. Webster said that the sum of evidence so far adduced, clinical, radiological, anatomical and experimental, rendered well-nigh irrefutable the thesis that the basis of epituberculosis so-called was a low-grade tuberculous pneumonia capable of resolution. But another conception of the origin of epituberculosis, apparently equally unassailable, was that of Terplan. That work, described in one of a series of morbid anatomical studies contributed to the *American Review of Tuberculosis*, sustained the contention that the clinical and radiological features which distinguished epituberculosis ensued on collapse of a pulmonary lobe or lobule brought about by occlusion of a major of minor bronchus. Such occlusion might be the result of pressure exerted on a bronchus by a large caseous lymph node, or might be determined by the irruption into the lumen of the bronchus of tuberculous granulation tissue or inspissated caseous material. With the healing of the morbid process or dispersal of its products, the lumen of the bronchus was restored and the collapsed portion of the lung expanded. The extent of atelectasis was directly related to the size of the occluded bronchus or bronchiole. Both those concepts of epituberculosis, the pneumonic and the atelectatic, seemed well reasoned and firmly founded, and it would probably be erroneous to adopt one explanation and ignore the other; indeed, it was not difficult to imagine circumstances in which both processes might operate in conjunction.

In conclusion, Dr. Webster stated that it was tempting to continue and discuss other questions relating to primary tuberculous infection, such as the evidence of a bacilliæmic phase, the nature and degree of enhanced resistance induced by primary infection, the unstable and fluctuating quality of such resistance, the concurrently developed hypersensitiveness which determined an acute inflammatory response to the injection of a minute amount of tuberculin, and the division of opinion as to whether or not the hypersensitive or allergic state contributed in any way to the operation or maintenance of acquired resistance. The weight of evidence seemed to indicate that it did not.

Radiological Aspects of Pulmonary Tuberculosis in Childhood.

DR. COLIN MACDONALD, speaking from the viewpoint of the radiologist, said that there was one important aspect of the subject which frequently remained unstated when a radiodiagnostic problem was being discussed, and that was the question of technique. Successful interpretation was dependent on successful technique, and few areas of the body required more attention to detail than the child's chest; there was much more to its radiography than the placing of the infant on a table, or the standing of an older child before an upright cassette and subsequently the pressing of a button. He held strongly to the view that it was better not to attempt any interpretation at all unless the films satisfied all the canons of good technique. For the radiologist, either in public hospital practice or in private, the quicksands of the Golden Pactolus were as solid rock compared with imperfect chest films, and the more tender the age, the more treacherous these became. Movement of heart and of diaphragm had to be overcome, postural symmetry had to be accomplished as far as was possible, and there must also be achieved that particular phase of respiration which was desired. Skill, patience and adequate assistance were required, and Dr. Macdonald's Celtic heart bled for the solitary technician struggling unaided with some of these devils incarnate.

Postural symmetry was most important and could not be achieved if the child was fighting like a tiger, or struggling like an eel. Dr. Macdonald did not dismiss the patients and their parents until he personally saw that the films were satisfactory, and so parents were warned that the examination might require up to an hour of their time. There should be no hesitation in repeating the films twice, or even thrice, if necessary. Interpretation was difficult enough in all conscience, without having to flounder in a morass of technical imperfections. That was one of the reasons, amongst many others, why in a public hospital it was essential to have a full-time radiologist on duty throughout the day, so that the patient did not leave the department until all the films had been passed as quite satisfactory. It took considerable experience for even keen and intelligent technicians to acquire proficiency in this difficult branch of radiography; indeed there were many, with the best will in the world, who never learnt how to handle young children.

Dr. Macdonald said that he avoided wherever possible interpretation from wet films. He knew there were occasions, numerous enough, when opinions had perforce to be given on wet films, but in the interest of clinician, radiologist and technician, he earnestly suggested that those occasions be kept at a minimum.

The radiographic prominence of the various components of the primary complex varied with the individual case; sometimes the adenomegaly was the only radiological clue. Yet many enlarged and even caseous glands escaped demonstration, as Dr. Reginald Webster had often pointed out. The lateral views were helpful in their demonstration, and that was one of the reasons why X-ray examination was quite incomplete with only an antero-posterior or postero-anterior view. As a routine, Dr. Macdonald always took both lateral views and frequently oblique ones. That thorough investigation was necessary to show small calcifications, and those were worth searching for, because far from being old, healed and of little account, they might be active foci of infection. The work of Dr. Douglas Galbraith, at the Orthopædic Hospital, Frankston, had shown how frequently calcified foci could explain a tuberculous spine or meningitis.

Tomography provided aid in the demonstration of the cavitation of caseous pneumonia, and Melbourne radiologists were indebted to Dr. Keith Hallam for showing the very considerable merit of tomography.

Dr. Macdonald did not share the marked antipathy of many eminent colleagues to the term "epituberculous". While he agreed that etymological objections to its use could be advanced, he thought that it possessed the merit of being a concise expression for a well-recognized clinico-radiological syndrome, of clinically benign but radiologically persisting, and eventually resolving, pulmonary consolidation. The lucid exposition given, a few minutes previously, by Dr. Webster of the varying pathogenesis of the syndrome should dispel any obscurantism in the matter. From the radiological aspect, it must be said that there was evidence that it was not always due to atelectasis.

Those who were interested in medical history, and who did not admit to any bedevilment by the employment of eponymous nomenclature, felt that a minor historical injustice had been committed by the linking of the primary focus with the name of Ghon alone. In point of fact, the real pioneer work on the complex was carried out some decades earlier by the Frenchman, Jules Marie Parrot, who first described the primary focus and its connexion with the bronchial glands in 1876, six years before Koch's discovery of the tubercle bacillus. Deriving from Parrot's work was that of Küss, who, in 1898, gave an excellent description of the primary complex, a term first used by the German, Ranke. Those investigations met with little interest on publication, and it was not until the Czech pathologist, Anton Ghon, published his monograph in 1912 that the subject was brought into the limelight. However, Ghon generously acknowledged his debt to Parrot and Küss, and so, if the focus was to be named after the physician who first recognized its importance, it should be called the "Parrot" focus. Parrot, who died in 1883 at the early age of forty-four years, belonged to the golden era of French clinical medicine. He was an acute observer, for it was he who first described the pseudo-paralysis of

congenital syphilitic osteochondritis, and also the skull nodes of congenital syphilis.

The diagnostic agent which the radiologist employed, namely, radiological inspection, often provided his clinical colleague with the greatest help; the mistakes that could be laid to its door were generally committed when the examination was too hurriedly, or too cheaply, carried out, or reports were written without due consideration being given to the clinical or pathological probabilities. He could give the audience a gentle assurance that nowadays radiologists were not shackled to a shadow. And speaking of shadows, Dr. Macdonald counselled moderation in the deprecation of those natural phenomena. It was the great English philosopher, John Locke, who contended that the picture of a shadow was a positive thing, and if one examined those same shadows more closely, they would be seen to be of the same order as the material things that caused them. And perhaps the thoughtful Wordsworth had something when he wrote that "we all laugh at pursuing a shadow, though the lives of the multitude are devoted to the chase".

In conclusion, Dr. Macdonald averred that it must not be thought that from his radiological glass-house, he would dare to dispatch any arrows at pathological or clinical shadows; he was a cautious Scot, and he saw in the audience much too formidable an array of dialectical talent.

Some Pulmonary Complications of Enlarged Tuberculous Mediastinal Glands.

DR. HOWARD WILLIAMS discussed the problem of some of the pulmonary complications that might occur from enlarged tuberculous mediastinal lymph nodes. He said that it would be strange if the enlarged glands, that were such a conspicuous and essential component of the primary pulmonary tuberculous complex, did not at times cause compression or ulceration of portion of the bronchial tree. Compression of portion of the bronchial tree or ulceration of the caseous glands into a bronchus might result in one, or a combination, of three pathological phenomena in almost any sequence: (i) emphysema from partial bronchial obstruction, (ii) collapse from complete bronchial obstruction, (iii) pneumonia from aspiration of infected material, either caseous tissue or tubercle bacilli. Healing of the caseous glands occurred slowly over a period of months or several years, and the bronchus which had been either compressed or ulcerated usually healed. At times there might be permanent bronchial stenosis from organization of the tuberculous granulation tissue. However, permanent changes might occur in the pulmonary tissue distal to the bronchial lesion, those changes being of the nature of either collapse with bronchiectasis or a caseating destructive pneumonic lesion.

Clinically, those pathological phenomena were usually manifested by widespread chronic pulmonary lesions of a segmental character in children who were not seriously ill. If there was radioopacity in the lung X-ray film then the syndrome was commonly referred to as "epituberculosis". It was often a source of considerable surprise to the clinician to find a large area of radioopacity in portion of the lung field when a relatively well-looking "Mantoux-positive" child was examined radiologically. Not infrequently there had been no symptoms of the nature of cough or stridor which might indicate that a lesion in the chest was present. Mild general ill health in a child who had been in contact with an adult with pulmonary tuberculosis was the only indication of such a lesion in the chest.

The lack of evidence as to the exact nature of the bronchial lesions in the group of children mentioned had been the factor responsible for confusion as to the exact pathology of epituberculosis. Bronchoscopic examination was the only certain means by which the evidence could be obtained. All degrees of narrowing of the bronchial lumen up to complete occlusion might be seen. Tuberculous granulation tissue might partially or completely plug the bronchial lumen and caseous material might be aspirated from the mediastinal lymph node or submucosal tuberculous lesion. It was a demonstration of pathological lesions of that type that explained the chronic segmental radiological shadows in the children under discussion and clearly demonstrated how pulmonary collapse might occur in one child and aspiration pneumonia in another. A sequence of events of the nature of segmental emphysema, collapse, then reexpansion was easily seen and demonstrated by bronchoscopic examination.

Dr. Williams then illustrated some of these clinical and pathological events with the case histories of three children.

The first child was a girl, aged two and a half years, whose primary infection had occurred at the age of approximately one year. Radiological examination at the age of fourteen months demonstrated enlarged left hilar lymph glands and, shortly afterwards, collapse of the major portion of the lower lobe of the left lung. After one year the lobe was still collapsed, the child during the whole of that time being entirely symptom-free and to all intents and purposes well, healthy and gaining weight. Bronchoscopic examination showed plugging of the left lower-lobe bronchus below the level of the dorsal-lobe bronchus with granulation tissue, culture of which demonstrated the presence of tubercle bacilli.

The second child was a little boy, aged two and a half years, who at the age of nine months became "off colour" and developed a cough. Examination disclosed a well-nourished, healthy looking child, who had clinical and radiological evidence of consolidation of the upper lobe of the left lung. Bronchoscopic examination disclosed compression of the left main bronchus from extrinsic pressure, and the lumen of the upper lobe of the left lung was completely occluded. Over the period of approximately ten months the shadow slowly resolved and all that remained was a little thickening and retraction of the lingula segment of the upper lobe. Tubercle bacilli were grown from the material aspirated from the left main bronchus. Examination by bronchogram one month before the meeting had demonstrated bronchiectasis and collapse in the lingula segment of the upper lobe of the left lung.

The third patient was a little girl who had been admitted to hospital at the age of six months with a history of wheezing and noisy breathing for a period of three months. It was thought that she might have inhaled a foreign body, as radiological and clinical examination demonstrated obstructive emphysema of the right lung. Bronchoscopic examination showed a considerable narrowing of the right main bronchus due to extrinsic pressure. The result of the child's Mantoux test was strongly positive, and culture of the fasting gastric content revealed the presence of tubercle bacilli. The child had been observed for a period of eighteen months and a most interesting sequence of pathological phenomena had been observed to occur in the right lung. After emphysema of the right lung occurred, collapse of the upper and middle lobes followed. Several months later reexpansion of the upper lobe of the right lung occurred with collapse of the lower lobe. The final result had been restoration of a normal bronchial lumen with normal upper and lower lobes in the right lung, but a collapsed middle lobe.

Discussion.

DR. A. P. DERHAM and DR. D. HAGGER both asked how infective was a child with a primary tuberculous complex and also if any member of the society had experience of a well-authenticated case of contact infection from child to child. The consensus of opinion of the meeting was that there was very little risk of contact infection from child to child, but occasionally a well-established case of such infection was seen. Bacteriological control must be the main criterion as to whether a child with a primary lesion was infectious or not.

DR. BELL FERGUSON said that there was less tuberculous infection in the community in Victoria at the present time than ten years previously. The falling death rate from tuberculosis in all age groups, the fewer children with tuberculous meningitis and the fewer patients with bone and joint tuberculosis all clearly established that statement.

DR. KATE CAMPBELL asked how reliable was the Vollmer patch test as compared with the Mantoux test in detecting tuberculous infection.

DR. K. G. KERR stated that in his experience it was quite reliable in children under the age of six years if carefully performed. In the group of children under discussion it was especially advantageous to use a patch test.

DR. ROBERT SOUTHBY said that he wished to draw attention to a syndrome occurring with tuberculous mastoiditis. Chronic otorrhœa, facial nerve palsy and enlarged glands on the side of the neck were the distinguishing features.

Correspondence.

THE PAINFUL STIFF SHOULDER.

SIR: Dr. Leigh T. Wedlick, in his article "The Painful Stiff Shoulder" (THE MEDICAL JOURNAL OF AUSTRALIA, November 12, 1949), speaks of adhesions in the shoulder joints. Although I am fully aware that most of the voluminous literature on the subject of stiff shoulder deals with adhesions as the cause of the limitation of shoulder movement, how many authors have actually seen them?

I have searched many articles and find them strangely silent about the actual appearance of these "adhesions".

Recently I have opened many shoulders at autopsies, all in the cases of sudden death, mostly of middle-aged and senile persons, and I have been unable to find anything resembling an adhesion in these joints. But I did find, what we know exists frequently, such lesions as partial tears of the supraspinatus tendon, thinning, flattening and rupture of the long tendon of the *biceps brachii*, corrosion of the bicipital groove and thinning of the subscapularis tendon—exactly those lesions which are recognized as potential causes of painful stiff shoulder.

I suggest the mechanism of painful stiff shoulder occurs in the following way. A minor injury occurs, such as a tear in the supraspinatus tendon, and this stimulates sensory nerve endings in this structure leading to contractions of the muscles appropriate for preventing excessive joint movement—that is, the adductors. Thus the arm is held at the side in adduction and internal rotation, aided by gravity. This protective contraction of the adductors can be quickly overcome by injection of local anæsthetic into the painful area and full abduction restored, at least temporarily, as Dr. Wedlick has stated. But if the adductors are allowed to remain in protective contraction for any length of time, the subscapularis and the lower part of the joint capsule probably become shortened and the abductors, especially the affected supraspinatus, become lengthened and abduction is limited.

The restoration of this abduction now requires drastic methods of treatment. The most drastic method is manipulation of the shoulder joint under general anæsthesia. Everyone who has carried out this procedure will verify the satisfactory noise and feeling which occur when abduction is produced, when "the adhesions are broken down". I think Codman was one of the first to realize exactly what occurs when a stiff shoulder is manipulated. He describes how he placed his finger into an open shoulder joint and at the same time he forced the arm into abduction; the fibres of the subscapularis were felt to part! Recently Nicola has reported two cases in which manipulation under general anæsthesia produced tears of the subscapularis tendon and anterior capsule of the joint.

In support of the theory that the lesion in stiff shoulder is shortening of the subscapularis and the lower part of the joint capsule, two further observations may be mentioned. First, it is rare to see an effusion in or around the joint after manipulation. Any blood or effusion produced by tearing the capsule would escape into the axilla and be absorbed. Secondly, after the manipulation a bruise may occasionally be seen above the medial epicondyle of the humerus—surely blood which has tracked down from a torn subscapularis or capsule.

I agree with Dr. Wedlick that many cases of stiff shoulder are preventible by early mobilization, and if I may be allowed to suggest an alteration to his aphorism "a movement a day keeps the adhesions away", it is "a movement a day keeps the spasm and shortening away".

Yours, etc.,

260 Saint George's Terrace, Perth, A. T. PEARSON.

November 28, 1949.

Post-Graduate Work.

THE MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

PROGRAMME FOR JANUARY-FEBRUARY, 1950.

THE following announcements are made by the Melbourne Permanent Post-Graduate Committee.

D.O. Part II.

The following courses suitable for candidates for the D.O. Part II will commence on Monday, January 16, 1950.

Lectures in pathology will be given on various days. Seven lectures will be given by Dr. Hugh Ryan at 12 noon in January at the Eye and Ear Hospital, and seven lectures by Dr. Kevin O'Day at 8 p.m. at Saint Vincent's Hospital in February. The fee will be 12 guineas.

Lectures in medical ophthalmology by Dr. G. Brew, Dr. A. Joyce and Dr. J. Billings, lectures in operative surgery by Dr. W. Box, and clinical lectures by the honorary staff of the Eye and Ear Hospital will be held on various days at the Eye and Ear Hospital. Clinical lectures and the lectures by Dr. Box, Dr. Brew and Dr. Joyce will be given at 4.30 p.m.; Dr. Billings's lectures will be given at 12 noon. The fee will be 17 guineas.

Lectures in bacteriology have been arranged by Professor S. D. Rubbo and will be given at the University of Melbourne at 4.30 p.m. on Mondays, Wednesdays and Fridays, commencing on February 1, 1950. The fee will be eight guineas.

Fees for Course.

Those enrolling for all the course for the D.O. Part II may do so for a fee of 35 guineas.

Bacteriology for D.G.O. and D.L.O.

Classes in bacteriology suitable for candidates for the D.G.O. and D.L.O. have been arranged by Professor S. D. Rubbo, and will be held at the University of Melbourne on Mondays, Wednesdays and Fridays at 4.30 p.m., commencing Wednesday, February 1, 1950. They will be completed before the diploma examinations on March 6, 1950. The fee will be eight guineas.

M.D. Part II and M.R.A.C.P.

A course in hæmatology suitable for candidates for the M.D. Part II and M.R.A.C.P. will be held at 2 p.m. on Tuesday and Thursday afternoons in February, 1950, as follows: February 2: introductory lecture by Dr. J. A. McLean, at the Royal Melbourne Hospital; February 7: "The Anæmias", by Dr. J. H. Bolton, at the Alfred Hospital; February 9: "Leuchæmia", by Dr. J. A. McLean, at the Royal Melbourne Hospital; February 14: "The Anæmias", by Dr. J. H. Bolton, at the Alfred Hospital; February 16: "Hæmorrhagic Disorders", by Dr. J. A. McLean, at the Royal Melbourne Hospital; February 21: "Spleen and Lymph Glands", by Dr. J. H. Bolton, at the Alfred Hospital. Each lecture will be followed by a clinical and laboratory demonstration. The fee will be £3 3s. for the course or 10s. 6d. per lecture.

Enrolments.

Enrolments for the above courses should be made with the Secretary of the Committee, 426 Albert Street, East Melbourne (telephone: JM 1547), as early as possible.

Country Courses.

Portland.

At Portland on Saturday, February 4, 1950, at 8.30 p.m., Dr. J. Eric Clarke will lecture on "Non-Tuberculous Pulmonary Disease". The fee will be 10s. 6d. Enrolments should be made with Dr. W. R. Angus, Koroit Street, Warrnambool. Telephone: 52.

Flinders Naval Depot.

At Flinders Naval Depot on Wednesday, February 8, 1950, at 2.30 p.m., Dr. B. Keon-Cohen will lecture on "Low Back Pain". This meeting is held by arrangement with the Royal Australian Navy.

Ballarat.

At Ballarat on February 18 and 19, 1950, the following lectures will be given: "Newer Aspects of Endocrinology", Dr. Keith Fairley; "Nephritis", Dr. D. G. Duffy; "Difficulties Encountered in Abdominal Surgery", Dr. W. E. Hughes-Jones; "Injuries of the Hand", Dr. A. R. Wakefield. The fee will be £2 2s. for the whole course or 10s. 6d. per lecture. Enrolments should be made with Dr. C. E. Richardson, Sturt Street, Ballarat. Telephone: Ballarat 1715.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

PROGRAMME FOR 1950.

General Revision Course.

THE Post-Graduate Committee in Medicine in the University of Sydney will be conducting its annual general revision course for two weeks beginning March 27, 1950. These courses will in future be held during the two weeks before Easter.

Annual Post-Graduate Oration.

The third post-graduate oration will be delivered in the Great Hall of the University of Sydney on the evening of Wednesday, March 29, 1950. The orator, Dr. S. A. Smith, will speak on "The Life and Work of Professor J. T. Wilson".

Those wishing to have their names placed on the invitation list are requested to communicate with the Secretary before February 1, 1950.

Special Courses.

The following dates, with the exception of those courses listed to begin in January and February, must be regarded as tentative only and are subject to confirmation.

Anæsthesia.—A six months' course suitable for candidates for the diploma in anæsthesia will begin on June 19, 1950.

Dermatology.—A course for candidates for Part I of the diploma in dermatology will begin on June 19, 1950, for a period of twelve weeks, followed by a Part II course in September covering a period of six months.

Gynæcology and Obstetrics.—A Part II course for candidates for the diploma in gynæcology and obstetrics, also suitable for M.R.C.O.G. candidates, will begin on January 9, 1950, for twelve weeks. A Part I diploma course will begin on August 28, 1950, for twelve weeks. Short courses in gynæcology and obstetrics will probably be held at the King George V Memorial Hospital for Mothers and Babies and at The Women's Hospital, Crown Street, during the latter half of the year. Post-graduate residencies at The Women's Hospital will be available from time to time, depending upon the number of vacancies.

Laryngology and Oto-Rhinology.—A Part II course for candidates for the diploma in laryngology and oto-rhinology will begin on January 9, 1950, for twelve weeks. A Part I course will begin on August 28, 1950, for twelve weeks.

Medicine.—Two courses in advanced medicine suitable for M.R.A.C.P. candidates will be held during the year for periods of fifteen weeks each, beginning on January 9 and May 29, 1950.

Ophthalmology.—A Part II course for candidates for the diploma in ophthalmology will begin on January 9, 1950, for twelve weeks. A Part I course will begin for twelve weeks on August 28, 1950.

Clinical Pathology.—A course for candidates for the diploma in clinical pathology will begin on July 10, 1950, for a period of six months.

Surgical Pathology.—A course of demonstrations in surgical pathology will be held during the month of October, 1950.

Psychological Medicine.—A Part I course for candidates for the diploma in psychological medicine will begin on March 20, 1950, for eight months. A Part II course will begin in June, 1950, for a period of twelve months.

Diagnostic Radiology.—A course for candidates for Parts I and II of the diploma in diagnostic radiology will begin on March 20, 1950. Arrangements can be made for candidates to be allocated over the full twelve months' period to recognized hospitals in order to qualify under the diploma regulations. Attendance is required full time over a period of twelve months unless candidates have already obtained the required experience.

Therapeutic Radiology.—A course for candidates for the diploma in therapeutic radiology will begin on March 20, 1950, during which candidates are allocated to recognized hospitals for a period of twelve months.

Pulmonary Tuberculosis.—A further week-end course in pulmonary tuberculosis will be conducted during the year, probably early in September.

Psychiatry.—During the year the committee proposes to conduct a course in psychiatry suitable for general practitioners on dates to be arranged.

Week-End Country Courses.

Courses will be held at the following country centres during the year: Armidale, March 4 and 5; Wagga Wagga, April 15 and 16; Albury, Katoomba, Broken Hill, June 10 and 11; Wollongong, July 29 and 30; Lismore, Taree, Parramatta, Newcastle, October 28 and 29; Bega, November 18 and 19; and Orange.

LIBRARY.

Books may be obtained on loan from the Post-Graduate Library by medical practitioners. The library includes reference books and books recommended for study for higher degrees and diplomas.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED DECEMBER 3, 1949.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[3]
Ankylostomiasis	*	5(5)	..	..	*	..	..	..	5
Anthrax	*	..	..	..	..	..	..	..	..
Beriberi	*	*	*	*	*	*	..	*	..
Bilharziasis	*	..	..	..	..	..	..	..	..
Cerebro-spinal Meningitis	..	5(2)	1(1)	..	..	..	..	..	6
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(*a*)	*	*	..	*	*	*	*	*	..
Dengue	*	*	*	*	*	*	..	*	..
Diarrhœa (Infantile)	..	..	7(4)	..	..	..	..	..	7
Diphtheria	7(5)	1	5(3)	1	5(2)	..	..	..	19
Dysentery(*b*)	*	1(1)	9(9)	..	..	..	..	..	10
Encephalitis Lethargica	..	..	..	..	..	..	..	..	..
Erysipelas	*	*	*	..	*	*	..	..	..
Filariasis	*	..	..	..	..	..	..	..	..
Helminthiasis	*	..	*	*	*	*	*	*	..
Hydatid	*	1	*	*	..	*	*	..	1
Influenza	*	*	*	..	*	*	..	*	..
Leprosy	..	..	..	..	..	..	..	..	..
Malaria(*c*)	*	(*c*)	(*c*)	(*c*)	(*c*)	(*c*)	(*c*)	(*c*)	(*c*)
Measles	*	*	*	51	*	*	..	..	51
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	6(2)	13(5)	..	58	..	5(3)	..	..	82
Psittacosis	*	..	*	..	*	*	*	*	..
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella(*h*)	*	*	2(2)	*	1(1)	1(1)	..	..	4
Scarlet Fever	47(14)	18(11)	4(2)	11	6(4)	..	..	..	86
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	*	..	*	*	*	*	*	*	..
Trachoma	*	*	*	*	*	*	*	*	..
Tuberculosis(*d*)	31(24)	18(12)	9(5)	5	14(12)	5	..	..	82
Typhoid Fever(*e*)	..	..	..	1	..	..	..	1	2
Typhus (Endemic)(*f*)	..	..	..	..	3(3)	..	..	..	3
Undulant Fever	*	*	*	*	*	*	*	*	..
Weil's Disease(*g*)	*	*	*	21	*	*	..	..	21
Whooping Cough	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 36, 1944–1945. Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from Northern Territory.
* Not notifiable.
(*a*) Includes "Mossman" and "Sarina" fevers. (*b*) Includes amœbic and bacillary. (*c*) Statistics inexact with varying practice with regard to relapses in Service cases infected overseas. (*d*) Includes all forms except in Northern Territory, where only pulmonary tuberculosis is notifiable. (*e*) Includes enteric fever, paratyphoid fevers and other Salmonella infections. (*f*) Cases reported include scrub, murine and tick typhus. (*g*) Includes leptospiroses, Weil's and para-Weil's disease. (*h*) Notifiable disease in Queensland in females aged over fourteen years.

OVERSEAS BUREAU.

All applications for post-graduate courses in England should be made through the committee. Difficulty is often encountered in regard to late applications and early notice must be given.

VISUAL AIDS DEPARTMENT.

Photographic Unit.—The photographic unit of the visual aids department can supply strip films, lantern and other slides; application should be made to the Secretary.

Film Library.—Films may be borrowed by various authorized bodies, and a loose-leaf catalogue containing details and appraisals of various films throughout Australia may be obtained at the cost of £1 1s.

INDIVIDUAL POST-GRADUATE CLINICAL STUDY.

Attendance at general or special clinics can be arranged with due notice to meet individual needs. Fees are £1 1s. for enrolment and £1 1s. per week.

INFORMATION.

All information concerning post-graduate matters may be obtained on application to the committee, 131 Macquarie Street, Sydney. Telegraphic and cable address: "Postgrad, Sydney." Telephones: BU 5238, BW 7483.

Australian Medical Board Proceedings.

QUEENSLAND.

THE following additional qualifications have been registered, pursuant to the provisions of *The Medical Acts*, 1939 *to* 1948, of Queensland.

Kurrle, Geoffrey Rosevear, M.D., 1949 (Univ. Melbourne), "Chelmsford", 33 Wickham Terrace, Brisbane.

Moni, Paul David Victor, D.Obst., R.C.O.G., 1948 (Univ. London), 49A Sandgate Road, Clayfield.

Martin, Norman William, Diploma of Ophthalmology, 1948 (Univ. Melbourne), No. 1 Wickham Terrace, Brisbane.

Nominations and Elections.

THE undermentioned have been elected as members of the New South Wales Branch of the British Medical Association:

Holt, Geoffrey Charles, M.B., B.S., 1947 (Univ. Sydney), 68 Allanan Street, Young.

Kalokerinos, James, M.B., B.S., 1948 (Univ. Sydney), 560 Old South Head Road, Rose Bay.

Stephens, Frederick Richard Neason, M.B., B.S., 1946 (Univ. Sydney), 49 New South Head Road, Vaucluse.

Fulde, Ewald Adolf Oscar, registered in accordance with the provisions of Section 17(2) of the *Medical Practitioners Act*, 1938-1945, 128 Brighton Boulevarde, North Bondi.

Corrigendum.

IN the "Current Comment" on "The Treatment of Status Epilepticus", published in the issue of November 5, 1949, at page 686, an error appears in line 11 of the second column. In this line "grammes" should read "grains", so that the sentence beginning on line 10 should read: "If phenobarbitone is used, it should be given in doses of six to twelve grains, preferably in the "soluble" form by the intravenous or intramuscular route." This error is regretted.

Medical Appointments.

Dr. A. L. Waddington has been appointed medical officer, Department of Public Health, New South Wales.

Dr. Robert Southby has been appointed a member of the Medical Board of Victoria, in pursuance of the provisions of Section 3 of the *Medical Act*, 1928, of Victoria.

Dr. R. D. Wright has been appointed a member of the Dietitians Registration Board, in pursuance of the provisions of Section 3 of the *Dietitians Registration Act*, 1942, of Victoria.

Diary for the Month.

JAN. 4.—Western Australian Branch, B.M.A.: Council Meeting.
JAN. 9.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JAN. 10.—New South Wales Branch, B.M.A.: Council Quarterly.
JAN. 12.—South Australian Branch, B.M.A.: Council Meeting.
JAN. 14.—Queensland Branch, B.M.A.: Council Meeting.
JAN. 16.—Federal Council of the B.M.A. in Australia: Meeting in Melbourne.
JAN. 17.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JAN. 25.—Victorian Branch, B.M.A.: Council Meeting.
JAN. 28.—Queensland Branch, B.M.A.: Council Meeting.
FEB. 1.—Western Australian Branch, B.M.A.: Council Meeting.
FEB. 2.—South Australian Branch, B.M.A.: Council Meeting.
FEB. 3.—Queensland Branch, B.M.A.: Branch Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135 Macquarie Street, Sydney): Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £3 *per annum* within Australia and the British Commonwealth of Nations, and £4 10s. *per annum* within America and foreign countries, payable in advance